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On the cover: A quillback rockfish, *Sebastes maliger*, photographed near Tenakee, Alaska, by Louis Barr of the NMFS Auke Bay Laboratory.

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A Summary of Tissue Lesions in Aquatic Animals Induced by Controlled Exposures to Environmental Contaminants, Chemotherapeutic Agents, and Potential Carcinogens

THEODORE R. MEYERS and JERRY D. HENDRICKS

Tissue lesions in fish and invertebrates induced by controlled exposures to various toxic compounds have been described in the literature. However, these reports are few in number, relative to other toxicological data, and widely scattered in several diverse journals. Therefore, retrieval of specific information regarding pathological changes caused by a particular compound(s) is a tedious and difficult task.

This article was written to provide a useful reference for aquatic toxicologists/pathologists by consolidating this descriptive material with key references through 1981. This information is presented in five tables. Table 1 identifies at least 90 environmental contaminants used as pesticides, herbicides, or as industrial chemicals which are categorized in the following manner: Organochlorines, petroleum compounds, organophosphates, carbamates, heavy metal salts, nitrogenous compounds, miscellaneous compounds, and chemotherapeutic agents. Table 4 lists at least 50 compounds grouped as mycotoxins, plant derivatives, and other chemicals all of which have been tested for carcinogenic activity. Tables 2, 3 and 5 describe and reference histologic and/or ultrastructural lesions or abnormalities caused by these compounds, their routes of exposure, and aquatic species tested.

Comparative discussions of lesions caused by these compounds are not intended here with the exception of the following comment. Most lesions induced by toxicant exposures are often indicative of toxic insult but nonspecific (i.e., hyperplastic, degenerative

Theodore R. Meyers, formerly with the Department of Food Science and Technology, Oregon State University, Corvallis, is currently with the School of Fisheries and Science, University of Alaska at Juneau, 11120 Glacier Highway, Juneau, AK 99801. Jerry D. Hendricks is with the Department of Food Science and Technology, Oregon State University, Corvallis, OR 97331. This work was supported in part by cooperative agreement (#CR809344-10) in the NCI/EPA Collaborative Program, Project No. 3, "Effects of Carcinogens, Mutagens and Teratogens in Nonhuman Species (Aquatic Animals)" administered by the Gulf Breeze Environmental Research Laboratory and Public Health Service Grants ES01926, ES00210, and ES00541 from the National Institute of Environmental Health Sciences. This is Technical Paper No. 6383, Oregon Agricultural Experiment Station, Oregon State Universit·. Mention of trade names or commercial products does not imply endorsement.

Table 1.—Organic and inorganic environmental contaminants and chemotherapeutic agents tested for ability to cause pathological changes in tissues of finfish and shellfish (see Tables 2 and 3)[1].

Organochlorine insecticides	Organochlorine herbicides	Industrial organochlorines	Petroleum compounds	Organophosphate insecticides	Carbamate insecticides	Miscellaneous herbicides	Metals and nitrogenous compounds
Chlordane	Dowpon	Chlorinated wastewater	Crude oil	Abate	Carbaryl (Sevin)	Acrolein	Ammonia (NH_3)
DDT	Dicamba	Polychlorinated biphenyls (PCB's)	Whole	Diazinon	Propoxur	Amitrole-T	Cadmium 109
Dieldrin	Dichlobenil	Aroclor 1248	Saltwater insoluble	Dimethoate		Atrazine	Cadmium chloride ($CdCl_2$)
Endosulfan	Diuron	Aroclor 1254	Saltwater soluble	Dursban		Dinoseb	Copper chloride ($CuCl_2$)
Endrin	Dowicide G	Miscellaneous PCB's	Waste motor oil	Dylox		Diquat	Copper nitrate ($CuNO_3$)
Heptachlor	Esteron	Carbon tetrachloride (CCl_4)	Oiled sediments	Fenthion		Hydrothol 191	Copper sulphate ($CuSO_4$)
Kepone	2,4-D		Oil slick	Malathion		Paraquat-CL	Landfill leachates
Lindane	Kuron (Silvex)		Miscellaneous hydrocarbons	Methyl Parathion		Sodium trichloroacetate ($NaTA_2$)	Lead acetate trihydrate ($C_6H_8O_6Pb$)
Methoxychlor	Kurosal		Miscellaneous hydrocarbons and PCB's			Trifluralin	Lead nitrate ($PbNO_3$)
Mirex	Tordon 101		Naphthalene				Mercuric chloride ($HgCl_2$)
Toxaphene	Tordon 22k		Phenol				Methyl mercury chloride (CH_3HgCl_2)
							Nitrite (NO_2)
							Silver chloride (AgCl)
							Sodium arsenate (Na_3HAsO_4)
							Sodium arsenite ($NaAsO_2$)
							Zinc chloride ($ZnCl_2$)
							Zinc sulphate ($ZnSO_4$)

Chemotherapeutic Agents		Other compounds	
Bradophen (disinfectant)	Neguvon, Masoten	Bis (tri-n-butyltin) oxide (molluscicide)	phosphoramide (TEPA-insect chemosterilant)
$CuSO_4$ (fungicide, algicide)	Ozone (biocide)	Dimethylsulfoxide (DMSO)	3-trifluoromethyl 4-nitrophenol (TFM-lampricide)
Dipterex	Penicillin G procain	Sodium lauryl sulfate (SLS-detergent)	Acid-alkaline pH (sulphuric acid; sodium hydroxide)
Emtrysidina	Dihydrostreptomycin sulfate	Triethylene	
Formalin (fungicide, ectoparasiticide)	Oxytetracycline		
Hexa-ex (disinfectant)	Sulfamethazine		
Kanamycin	Sulfonamides		
Malachite Green (fungicide, ectoparasiticide)	Thiabendazole (vermicide)		
Methylene Blue (fungicide, disinfectant)	Tobramycin		
	Yomesan (anthelminthic)		

[1]Source: Figure 1 (Meyers and Hendricks, in press) in G. M. Rand and S. Petrocelli (editors), "Principles of aquatic toxicology," Hemisphere Publishing Corporation, N.Y.

Table 2.—Summary of reported gross, histological, and ultrastructural changes in tissues of fish and shellfish resulting from exposures to various chemical compounds. Descriptions and interpretations are provided mostly in the words of the authors cited which may be vague or ambiguous in some cases. For "Exposure routes," W = water, D = diet, IP = intraperitoneal injection, and GI = intragastric intubation. Contaminant abbreviations are defined in Table 1.

Contaminant	Exposure route	Species	Tissues affected	Lesions
Organochlorine insecticides				
Chlordane	W	Lake trout (*Salvelinus namaycush*)	Liver	Increased amounts of focal hepatocyte vacuolation and degeneration (Eller, unpubl., in Couch, 1975).
DDT	W	Coho salmon (*Oncorhynchus kisutch*)	Integument	Erosion of premaxillary region[1] (Walsh and Ribelin, 1975).
			Gills	Respiratory epithelial hyperplasia of secondary lamellae[1] (Walsh and Ribelin, 1975).
			Spleen	Hypocellularity and fibroplasia (Walsh and Ribelin, 1975).
			Liver	Hyperemia, petechiae, fatty change, periportal necrosis, disorganized architecture (Walsh and Ribelin, 1975).
			Intestine	Hyperemia, lymphocytic infiltration (Walsh and Ribelin, 1975).
			Brain	Hyperemia or hemorrhage (Walsh and Ribelin, 1975).

and/or necrotic) changes making diagnosis of a single causative compound or group of compounds difficult, if not impossible. Only a limited number of toxicants cause lesions in aquatic species which may be useful for diagnostic purposes, although none are pathognomonic; carbaryl causes muscular and neural lesions (Walsh and Ribelin, 1975) while varying degrees of vertebral deformities are caused by kepone (Couch et al., 1977), toxaphene (Mehrle and Mayer, 1975; Mayer et al., 1978), trifluralin (Couch et al., 1979), malathion (Mount and Stephan, 1967; Weis and Weis, 1976), various organophosphates (McCann and Jasper, 1972), carbamates (Carter, 1971), and lead salts (Holcombe et al., 1976; Ozoh 1979a,b). For detailed discussions of these and other less specific lesions the investigator is referred to Meyers and Hendricks (in press) or the appropriate literature sources listed. A discussion of carcinogens and neoplastic lesions in fish is presented by Hendricks (1982).

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Table 2.—Continued.

Contaminant	Exposure route	Species	Tissues affected	Lesions
DDT (cont.)	W	Lake trout	Gills	Respiratory epithelial hyperplasia of secondary lamellae[1] (Walsh and Ribelin, 1975).
			Spleen	Paleness, atrophy, hypocellularity, fibroplasia (Walsh and Ribelin, 1975).
			Liver	Hyperemia, petechiae, fatty change (Walsh and Ribelin, 1975).
			Intestine	Air distension in fry (Burdick et al., 1964), hyperemia and lymphocytic infiltration (Walsh and Ribelin, 1975).
			Swim bladder	Air distension in fry (Burdick et al., 1964).
			Brain	Hyperemia or hemorrhage (Walsh and Ribelin, 1975).
	D	Chinook salmon (*O. tshawytscha*)	None	(Buhler et al., 1969).
	W	Cutthroat trout (*Salmo clarki*)	None	(Allison et al., 1964).
	D & W	Rainbow trout (*S. gairdneri*)	None	(Wood, unpubl., in Walsh and Ribelin, 1975).
			Liver	Hepatic cell carcinoma (Halver et al., 1962).
	W	Brown trout (*S. trutta*)	Liver	Nuclear hypertrophy and cytoplasmic vacuolation of hepatocytes (King, 1962).
			Kidney	Tubular degeneration and debris in lumina (King, 1962).
			Intestine	Submucosal vacuolization and degeneration of mucosal epithelium (King, 1962).
			Adrenal cortex (interrenal cells)	Necrosis (King, 1962).
	W	Guppy (*Poecilia reticulata*)	Liver	Severe necrosis (King, 1962).
			Adrenal cortex (Interrenal cells)	Necrosis (King, 1962).
			Spleen	Atrophy (King, 1962).
	W	Asian fish sp. (unspecified)	Liver	Hepatocyte hypertrophy, degeneration, necrosis (Mathur, 1962).
			Kidney	Tubular degeneration (Mathur, 1962).
			Intestine	Submucosal vacuolization, degeneration of mucosal epithelium, loss of goblet cells (Mathur, 1962).
	W	Eel (*Anguilla rostrata*)	Intestine	ATPase inhibition (Janicki and Kinter, 1971).
			Pituitary	Degranulation of ε cells (DDD) (Ball and Baker, 1969).
DDT, Toxaphene and Parathion (mixture)		American oyster† (*Crassostrea virginica*)	Digestive diverticula (Smaller nonciliated tubules)	Necrosis of tubular epithelium, reduction in tubule height and dilation of lumina (Lowe et al., 1971b).
			Gonads	Inhibition of maturation in female oysters with leukocyte infiltration or hyperplasia of germinal epithelium; leukocyte infiltration among spermatozoa in males (Lowe et al., 1971a,b).
			Vesicular connective tissue	Edema beneath intestinal areas sometimes with infiltration of leukocytes (Lowe et al., 1971b).
			Mantle, gut, gonads, gills, visceral ganglion & kidney tubules	Secondary infection with a mycelial fungus causing cell lysis, usually without host leukocyte infiltration (Lowe et al., 1971b).
Dieldrin	W	Various fishes	Liver	Hepatocyte pleomorphism, cytoplasmic vacuoles (Mathur, 1965).
	W	Lake trout	Gills	Hyperplasia of lamellar respiratory epithelium[1] and telangiectasia (Walsh and Ribelin, 1975).
			Spleen	Paleness, atrophy, hypocellularity[1], and fibroplasia (Walsh and Ribelin, 1975).
			Liver	Hyperemia, petechiae or discolored areas, fatty change, congestion of sinusoids and hepatic veins (Walsh and Ribelin, 1975).
			Intestine	Hyperemia (Walsh and Ribelin, 1975).
			Brain	Hyperemia[1] or hemorrhage (Walsh and Ribelin, 1975).
	W	Coho salmon	Integument	Erosion of premaxillary region[1] (Walsh and Ribelin, 1975).
			Gills	Hyperplasia of lamellar respiratory epithelium[1] and telangiectasia (Walsh and Ribelin, 1975).
			Spleen	Hypocellularity[1] and fibroplasia (Walsh and Ribelin, 1975).
			Liver	Hyperemia, petechiae, or discolored areas, fatty change, congestion of sinusoids and hepatic veins (Walsh and Ribelin, 1975).
			Intestine	Hyperemia (Walsh and Ribelin, 1975).
			Brain	Hyperemia or hemorrhage (Walsh and Ribelin, 1975).
			Adrenal cortex (Interrenal cells)	Hyperplasia (Walsh and Ribelin, 1975).
	W	Asian fish sp. (*Ophiocephalus punctatus*)	Liver	Hypertrophy of hepatocytes, vacuolar degeneration of cytoplasm and necrosis (Mathur, 1975).
		(*Trichogaster fasciatus*)	Liver	Vacuolar degeneration of hepatocytes and localized necrosis (Mathur, 1975).
Endosulfan	W	Lake trout	Gills	Telangiectasia and hyperplasia[1] of lamellar respiratory epithelium (Walsh and Ribelin, 1975).
			Spleen	Paleness, atrophy, hypocellularity, fibroplasia (Walsh and Ribelin, 1975).
			Liver	Petechiae or discoloration, fatty change (Walsh and Ribelin, 1975).
			Intestine	Hyperemia (Walsh and Ribelin, 1975).
			Brain	Hyperemia[1] or hemorrhage (Walsh and Ribelin, 1975).
	W	Coho salmon	Integument	Erosion of premaxillary region[1] (Walsh and Ribelin, 1975).
			Spleen	Paleness, atrophy, hypocellularity, and fibroplasia (Walsh and Ribelin, 1975).
			Liver	Petechiae or discolored areas, fatty change (Walsh and Ribelin, 1975).
			Intestine	Hyperemia (Walsh and Ribelin, 1975).
			Brain	Hyperemia or hemorrhage (Walsh and Ribelin, 1975).
Endrin	W	Rainbow trout	Liver	Hepatocyte degeneration (Wood, unpubl., in Couch, 1975).
			Kidney	Yellow pigment in tubules (Wood, unpubl., in Couch, 1975).
	D & W	Cutthroat trout	Gills	Edema and separation of respiratory epithelium from basement membrane (Eller, 1971).
			Liver	Suggestive preneoplastic changes which never developed into hepatic cell carcinoma (Eller, 1971).
			Ovary	Hyperplasia of germinal epithelium and involution of some follicles (Eller, 1971).
			Pancreas	Islet cell hyperplasia (Eller, 1971).
	W	Spot (*Leiostomus xanthurus*)	Liver	Focal necrosis of hepatocytes with inflammation and loss of glycogen and lipid (Lowe, 1965).

†Note: Lesions were absent in oysters exposed to separate concentrations of each compound (Lowe et al., 1971b).

Table 2.—Continued.

Contaminant	Exposure route	Species	Tissues affected	Lesions
Endrin (cont.)	W	Spot (cont.)	None	(Johnson, 1968).
		Guppy (*P. reticulata*)	Liver	Fatty change (Mount, 1962).
	W	Fish (sp. not given)	Kidney	Vacuolated cells in glomeruli (Mount and Putnicki, 1966).
	W	Goldfish	Liver	Reduced cytoplasmic vacuolization (Grant and Mehrle, 1970).
		(*Carassius auratus*)	Thyroid	Reduced follicular cell height (Grant and Mehrle, 1970).
	IP	Asian catfish	Liver	Hypertrophy of hepatocytes and hepatocyte nuclei, centrilobular necrosis, perilobular vacuolization, fibrosis (Sastry and Sharma, 1978).
		(*Channa punctatus*)		
Heptachlor	W	Rainbow trout	Gills	Edema and congestion (Wood, unpubl., in Couch, 1975).
			Liver	Heavy bile pigment deposits (Wood, unpubl., in Couch, 1975).
	W	Cutthroat trout	Gills	Edema and congestion (Andrews et al., 1966).
			Liver	Hepatocyte degeneration and deposition of bile pigments (Andrews et al., 1966).
	D & W	Bluegill	None	(Cope, 1963).
		(*Lepomis macrochirus*)	Liver	Hepatocyte shrinkage with loss of lipid and glycogen, loss of normal architecture (Andrews et al., 1966).
			Peripheral blood	Enlarged erythrocytes (Andrews et al., 1966).
Kepone	W	Sheepshead minnow	Vertebral column	Scoliosis, fractured centra of vertebrae with foci of osteoblastic repair (Couch et al., 1977).
		(*Cyprinodon variegatus*)	Spinal cord	Compression of lateral funiculi by vertebral fractures (Couch et al., 1977).
			Skeletal muscle	Distorted myotomal patterns, loss of neurological control of caudal melanocyte pattern (Couch et al., 1977).
Lindane	W	Rainbow trout	Liver	Focal necrosis (Wood, unpubl., in Walsh and Ribelin, 1975).
			Kidney	Degeneration of tubules (Wood, unpubl., in Walsh and Ribelin, 1975).
	W	Asian fish sp.	Liver	Vacuolar degeneration of hepatocytes with slight to moderate necrosis and atrophy with loss of normal cord pattern (Mathur, 1975).
		(*O. punctatus*)		
		(*T. fasciatus*)	Liver	Cytoplasmic alterations of hepatocytes, margination of nuclear chromatin and hypertrophy (Mathur, 1975).
Methoxychlor	W	Rainbow trout	Liver	Nonspecific degeneration (Wood, unpubl., in Walsh and Ribelin, 1975; Cope, 1966).
	W	Bluegill	Liver	Shrinkage and granulation of hepatocytes, loss of normal cord pattern, eosinophilic globules in capillary lumina (Kennedy et al., 1970).
			Brain	Vascular congestion (Kennedy et al., 1970).
	W	Carp fry	Gills	Increased mucus secretions, respiratory epithelial hyperplasia with "clubbing" and fusion of lamellae, necrosis and desquamation of lamellar respiratory epithelium and vascular congestion or telangiectasia (Lakota et al., 1978).
		(*Cyprinus carpio*)		
			Liver	Vascular congestion and hepatocyte degeneration (Lakota et al., 1978).
			Kidney	None (Lakota et al., 1978).
			Intestine	None (Lakota et al., 1978).
	W	Mummichog	Lateral line canal	Epithelial necrosis (Gardner, 1975).
		(*Fundulus heteroclitus*)		
Mirex	W	Cutthroat trout	Gills	Fused lamellae (Van Valin et al., 1968).
	W	Goldfish	Gills	Edema and telangiectasia of lamellae (Van Valin et al., 1968).
			Gallbladder	Distended with flattened epithelium (Van Valin et al., 1968).
	W	Bluegill	None	(Van Valin et al., 1968).
	D	Pinfish		
		(*Lagodon rhomboides*)	None	(Lowe et al., 1971a).
Toxaphene	W	Rainbow trout	Gills	Edema (Wood, unpubl., in Walsh and Ribelin, 1975).
			Liver	Hepatocyte necrosis and disorganization of cord architecture (Wood, unpubl., in Walsh and Ribelin, 1975).
	W	Fathead minnow	Spinal vertebrae	Reduced collagen content and hyperfragility (Mehrle and Mayer, 1975).
		(*Pimephales promelas*)		
	W	Spot	Gills	Thickening of lamellar respiratory epithelium (Lowe, 1964).
	D	Channel catfish	Integument	Reduction in mucus cell numbers and increased thickness of epidermis (Mayer et al., 1978).
		(*Ictalurus punctatus*)		
			Spinal vertebrae	Causes a functional vitamin C deficiency resulting in reduction of vertebral collagen and spinal deformities (Mayer et al., 1978).
Organochlorine herbicides				
Dowpon	W	Carp	Pancreas	Submicroscopic degeneration to acute necrosis of acinar cells (Schulz, 1971).
Dicamba	W	Coho salmon	None	(Hendricks, 1979).
Dichlobenil	W	Bluegill	Gills	Vascular engorgement with lamellar telangiectasia and fusion (Cope, 1966).
			Liver	Hepatocyte necrosis characterized by pyknosis and karyolysis, fibrosis and adenomatous change (Cope et al., 1969).
Diuron	W	Bluegill	Gills	Lamellar telangiectasia, hemorrhage and respiratory epithelial hyperplasia with fusion (McCraren et al., 1969).
Dowicide G	W	Guppy	Liver	Enlarged sinusoids, hypertrophied, hyperchromic hepatocyte nuclei, less lipid deposition, hepatocyte necrosis in one fish and fatty change in another (Crandall and Goodnight, 1963).
Esteron	W	Coho salmon	Gills	Curving of filaments, exfoliation of some chloride cells and congestion of most lamellae resulting in telangiectasia in some (Hendricks, 1979).
2,4-D	W	Bluegill	Liver	Hepatocyte shrinkage, loss of glycogen, distortion of hepatic cords, atypical hepatocyctes (Cope et al., 1970).
			Brain	Vascular congestion (Cope et al., 1970).
			Blood vessels	PAS positive globular masses in lumina (Cope et al., 1970).
	W	Lake trout	Gills	Lamellar respiratory epithelial hyperplasia[1], telangiectasia (Walsh and Ribelin, 1975).
			Spleen	Hypocellularity[1], fibroplasia (Walsh and Ribelin, 1975).
			Liver	Hyperemia, fatty change[1] and congestion of sinusoids and veins (Walsh and Ribelin, 1975).
			Bile	Pink or red color (Walsh and Ribelin, 1975).
			Intestine	Hyperemic, flaccid (Walsh and Ribelin, 1975).
			Brain	Hyperemia or hemorrhage (Walsh and Ribelin, 1975).
	W	Coho salmon	Gills	Hyperplasia of lamellae[1] (Walsh and Ribelin, 1975).
			Spleen	Hypocellularity, fibroplasia (Walsh and Ribelin, 1975).
			Liver	Hyperemia, fatty change, congestion of sinusoids and veins (Walsh and Ribelin, 1975).

Table 2.—Continued.

Contaminant	Exposure route	Species	Tissues affected	Lesions
2,4-D (cont.)	W	Coho salmon (cont.)	Bile	Pink or red color (Walsh and Ribelin, 1975).
			Intestine	Hyperemic, flaccid (Walsh and Ribelin, 1975).
			Brain	Hyperemia or hemorrhage (Walsh and Ribelin, 1975).
Kuron (Silvex)	W	Bluegill	Liver	Hepatocyte shrinkage, loss of glycogen, distortion of cord architecture (Wood, unpubl., in Walsh and Ribelin, 1975).
			Testes	Stimulation of spermatogenesis and exhaustion atrophy (Wood, unpubl., in Walsh and Ribelin, 1975).
Kurosal	W	Bluegill	None	(Wood, unpubl., in Walsh and Ribelin, 1975).
Tordon 101	W	Coho salmon	Liver	Peribiliary necrosis (Hendricks, 1979).
Tordon 22k	W	Coho salmon	Liver	Some hydropic degeneration and some hypertrophy of hepatocytes containing fiber-like strands in the cytoplasm (Hendricks, 1979).
			Gills	Some hypertrophy of respiratory epithelial cells while others had a shrunken appearance (Hendricks, 1979).
Industrial organochlorines				
Chlorinated wastewater	W	Coho salmon	Peripheral blood	Microcytic, hypochromic hemolytic anemia with high percentage of circulating abnormal and immature red blood cells (Buckley et al., 1976); formation of Heinze bodies and Howell-Jolly bodies (Buckley, 1977).
PCB's				
Aroclor 1248	W	Lake trout	Liver	Focal hepatocyte degeneration, cytoplasmic vacuolation and pleomorphism more severe than in control fish (Eller, unpubl., in Couch, 1975).
Aroclor 1254	W	Spot	Liver	Increased fatty change, vacuolation and necrosis of pancreatic acinar tissue around portal tracts with infiltration of lymphocytes, focal hepatocyte necrosis, sinusoidal congestion and presence of ceroid-like inclusion bodies in parenchymal cytoplasm (Couch, 1975).
	D	Rainbow trout	Kidney	Degeneration of renal tubules characterized by flattening and pyknosis of the epithelium, dilation of the lumina with proteinaceous casts and desquamated epithelial cells (Nestel and Budd, 1975).
			Liver	Variable degree of vacuolation and hepatocyte density of questionable significance (Nestel and Budd, 1975); distinct vacuolation of hepatocytes and enlargement of rough ER no longer adjacent to nuclei or mitochondria (Sivarajah et al., 1978).
			Spleen	Less pigment (melanin?) and red pulp per unit area and reduced amounts of white pulp (Nestel and Budd, 1975).
	IP		Testes	Ultrastructural damage to spermatozoa particularly in head region — loss of serrations in outer membrane (Sivarajah et al., 1978).
			Ovaries	Fragmented cytoplasm in oocytes due to enlargement and proliferation of smooth ER (Sivarajah et al., 1978).
	IP	Carp	Liver	Enlargement of rough ER (Sivarajah et al., 1978).
			Testes	Ultrastructural damage to spermatozoa particularly in head region — loss of serrations in outer membrane (Sivarajah et al., 1978).
			Ovaries	Fragmented cytoplasm in oocytes due to enlargement and proliferation of smooth ER (Sivarajah et al., 1978).
	W	American oyster††	Digestive diverticula	Atrophy of tubular epithelium characterized by cytoplasmic vacuolation, reduction in height and dilation of lumina (Lowe et al., 1972).
			Vesicular connective tissue	Loss of compact architecture (edema) with heavy infiltration of leukocytes (Lowe et al., 1972).
			Gonads	Leukocyte infiltration (Lowe et al., 1972).
Miscellaneous PCB's	D	Chinook salmon	Intestine	Exfoliation of mucosal epithelium, absence or reduction in brush border and presence of abnormal cytoplasmic inclusion bodies in columnar epithelium (Hawkes et al., 1980).
			Liver	Vesiculated rough endoplasmic reticulum and circular arrays of smooth surfaced membranes and myelin-like bodies in hepatocyte cytoplasm (Hawkes, 1980).
	GI	Channel catfish	Liver	Proliferation of endoplasmic reticulum and "bizarre" whorls of both rough and smooth ER (Hinton et al., 1978; Klaunig et al., 1979).
CCl_4	IP	Rainbow trout	Liver	Vacuolation of hepatocytes and compression of sinusoids with eventual hepatocyte necrosis (Racicot et al., 1975); mottled with blanched areas, eosinophilic degeneration and hydropic degeneration of hepatocytes, pyknosis and coagulative necrosis in subcapsular areas while liquefactive necrosis and karyolysis predominated in centrilobular regions (Gingerich et al., 1978).
			Peritoneum	Dark red serous ascites and hemorrhagic inflammation (Gingerich et al., 1978).
			Intestine	Hemorrhagic inflammation and multiple thrombi in ventral intestinal vein (Gingerich et al., 1978).
			Spleen	Mottled with blanched areas (Gingerich et al., 1978).
Petroleum compounds				
Crude oil Whole	W	Atlantic silverside (*Menidia menidia*)	Olfactory organs	Hyperplasia of sustentacular epithelium, cellular degeneration and necrosis of olfactory mucosa (Gardner, 1975).
			Heart	Degeneration of ventricular myocardium (Gardner, 1975).
			Pseudobranch	Degeneration of secretory cells (Gardner, 1975).
	W	Pacific herring (embryos) (*Clupea pallasii*)	Anterior brain	Enlarged and irregularly shaped nonmembrane bound perinuclear and intracellular spaces (Cameron and Smith, 1980).
			Skeletal muscle	Numerous swollen mitochondria with some disruption of internal membranes and cristae; intercellular breakdown of membranes (Cameron and Smith, 1980).
	D	Rainbow trout	Liver	Loss of glycogen reserves in hepatocytes, proliferation of endoplasmic reticulum and presence of cochlear ribosomes, fibrosis around sinusoids (Hawkes, 1977).
			Eye lens	Enlarged, abnormally soft, probably resulting from shriveling and degeneration of lens fibers observed by TEM (Hawkes, 1977).
Saltwater insoluble	W	Atlantic silverside	Olfactory organs	Hyperplasia of sustentacular epithelium and necrosis, vasodilation of submucosa (Gardner, 1975).
Saltwater soluble	W	Atlantic silverside	Olfactory organs	Epithelial metaplasia of olfactory mucosa with appearance of extracellular refractile rods; some hyperplasia and cytoplasmic degeneration in neurosensory and sustentacular epithelium in basal region of olfactory lamellae (Gardner, 1975).

††Note: Lesions reversible following 12 weeks depuration in PCB-free seawater (Lowe et al., 1972).

Table 2.—Continued.

Contaminant	Exposure route	Species	Tissues affected	Lesions
Crude oil (cont.)				
Saltwater soluble (cont.)	W	Coho salmon; starry flounder (*Platichthys stellatus*)	Gills	Sloughing of first 2-3 layers of surface respiratory epithelial cells with excessive mucus exudation (Hawkes, 1977).
	W	English sole (*Parophrys vetulus*)	Integument	Increased numbers of mucus glands which appeared dilated when observed with SEM (Hawkes, 1977).
	W	Sand sole (*Psettichthys melanostictus*)	Nares epithelium	Severe damage to receptor organelles including degeneration of chemosensory cilia and loss of microridges circumscribing epithelial cells surrounding olfactory organs (Hawkes, 1980).
Waste motor oil	W	Atlantic silverside	Blood vessels	Vascular anomalies (Gardner, 1978).
Oiled sediments	W	English sole	Liver	Increase in lipid volume per hepatocyte (McCain et al., 1978).
Oil slick	W	Cunner (*Tautogolabrus adspersus*)	Eye lens	Degeneration of lens fibers (Payne et al., 1978).
Miscellaneous hydrocarbons	D	Chinook salmon	Intestine	Abnormal inclusion bodies in apical cytoplasm of mucosal columnar epithelium, cytoplasmic vesiculation in columnar cells near luminal surface and abnormal electron lucent cytoplasm and increased amounts of granular endoplasmic reticulum in basal mucosal cells (Hawkes et al., 1980).
Miscellaneous hydrocarbons and PCB's	D	Chinook salmon	Intestine	Increased exfoliation of mucosa, reduced brush border, diffuse distribution of mitochondria in cytoplasm, changes in vesiculation of cytoplasm near brush border, cytoplasmic inclusion bodies throughout cell, increased endoplasmic reticulum and reduction in cytoplasmic density (Hawkes et al., 1980).
Naphthalene	W	Mummichog	Gills	Hemorrhage at base of filaments and respiratory epithelial hyperplasia (DiMichele and Taylor, 1978).
			Neurosensory organs	Degeneration and necrosis of sensory epithelium and sustentacular cells (DiMichele and Taylor, 1978).
			Pancreas; liver; kidney; brain	Vascular congestion and infarction (DiMichele and Taylor, 1978).
			Adrenal cortex (interrenal cells)	Hypertrophy and necrosis (DiMichele and Taylor, 1978).
			Intestine	Necrosis and sloughing of mucosa (DiMichele and Taylor, 1978).
			Skeletal muscle	Necrosis (DiMichele and Taylor, 1978).
Phenol	W	Bream (*Abramis brama*)	Gills	Respiratory epithelial necrosis, extravasation of blood in lamellae (Waluga, 1966b).
			Integument	Increased numbers of mucus glands which were distended (Waluga, 1966a).
			Kidney	Gross discoloration with congestion of vessels (Waluga, 1966a).
	W	14 assorted fishes	Gills	Discharged mucus glands and generalized inflammation (Reichenbach-Klinke, 1965).
			Integument	Swollen epidermis and generalized inflammation (Reichenbach-Klinke, 1965).
	W	Rainbow trout	Gills; pharynx	Necrosis and erosion of epithelium with generalized inflammation of gills (Mitrovic et al., 1968).
	W	Hard clam (*Mercenaria mercenaria*)	Gills	Respiratory epithelial necrosis and desquamation, distension of hemolymph sinuses containing an amorphous precipitate (Fries and Tripp, 1976).
			Intestine	Epithelial necrosis and sloughing (Fries and Tripp, 1976).
			Digestive diverticula	Tubular epithelial necrosis and sloughing (Fries and Tripp, 1976).
Organophosphate insecticides				
Abate	W	Bluegill	Liver	Atrophy distortion of muralia, variability in stain affinity of hepatocytes, large foci of edema, congestion and hepatocyte necrosis (Eller, unpubl. in Couch, 1975).
			Pancreas	Acinar cell necrosis (Eller, unpubl. in Couch, 1975).
Diazinon	W	Asian catfish	Intestine	Cytoplasmic vacuolation and loss of structural integrity of mucosal folds, granular inclusions in mucosal and submucosal regions, dilation of submucosal vasculature, necrosis of submucosa, and degeneration of intestinal smooth muscle layers (Anees, 1976).
			Liver; kidney	Granular dystrophy and cytoplasmic vacuolation (Anees, 1976).
Dimethoate	W	Asian catfish	Intestine	Moderate cytoplasmic vacuolation of villi extremities, vascular dilation and necrosis in submucosa and slight degeneration of smooth muscle layers (Anees, 1976).
			Liver; kidney	Granular dystrophy and cytoplasmic vacuolation (Anees, 1976).
Dursban	W	Sheepshead minnow	Liver	Congestion, fatty change secondary to change in food consumption (Lowe, pers. commun., in Couch, 1975).
Dylox	W	Rainbow trout	Liver	Vacuolation in cytoplasm of hepatocytes (Matton and LaHam, 1969).
Fenthion	W	Cichlids (*Herotilapia multispinosa*) (*Tilapia leucosticta*)	Gills	Hyperplasia and separation of lamellar respiratory epithelium from basement membranes, telangiectasia, and thombosis (Jauch, 1979).
			Gills	Severe respiratory epithelial hyperplasia and lamellar fusion (Jauch, 1979).
Malathion	W	Rainbow trout	Gills	Edema and respiratory epithelial separation (Wood, unpubl. in Walsh and Ribelin, 1975).
			Liver	Nonspecific degeneration (Wood, unpubl. in Walsh and Ribelin, 1975).
	W	Cutthroat trout	None	(Allison et al., 1964).
	W	Lake trout	Fins	Hemorrhage at base of dorsal fin (Walsh and Ribelin, 1975).
			Gills	Hyperplasia of lamellar respiratory epithelium[1] (Walsh and Ribelin, 1975).
			Spleen	Hypocellularity[1] and fibroplasia (Walsh and Ribelin, 1975).
			Liver	Fatty change[1] (Walsh and Ribelin, 1975).
			Intestine	Hyperemia (Walsh and Ribelin, 1975).
			Brain	Hyperemia or hemorrhage (Walsh and Ribelin, 1975).
	W	Coho salmon	Integument	Erosion of premaxillary region[1] (Walsh and Ribelin, 1975).
			Gills	Hypertrophy and hyperplasia[1] of lamellar respiratory epithelium, telangiectasia (Walsh and Ribelin, 1975).
			Spleen	Hypocellularity[1] and fibroplasia (Walsh and Ribelin, 1975).
			Liver	Fatty change[1] and glycogen deposits (Walsh and Ribelin, 1975).
			Gall bladder	Alteration of epithelium (Walsh and Ribelin, 1975).
			Intestine	Hyperemia (Walsh and Ribelin, 1975).
			Brain	Hyperemia or hemorrhage (Walsh and Ribelin, 1975).
Methyl Parathion	W	Rainbow trout	Gills	Marked respiratory epithelial hyperplasia (Wood, unpubl. in Walsh and Ribelin, 1975).
			Liver	Swelling of hepatocytes and sinusoid congestion (Wood, unpubl. in Walsh and Ribelin, 1975).

Table 2.—Continued.

Contaminant	Exposure route	Species	Tissues affected	Lesions
Methyl Parathion (cont.)	W	Rainbow trout (cont.)	Kidney	Proteinaceous casts in Bowman's space and lumina of collecting tubules (Wood, unpubl. in Walsh and Ribelin, 1975).
	W	Asian catfish	Intestine	Necrotic mucosa, dilated submucosal vasculature, and irregular pattern of cellular structures (Anees, 1976).
			Liver; kidney	Granular dystrophy and cytoplasmic vacuolation (Anees, 1976).
Carbamate insecticides				
Carbaryl (Sevin)	W	Rainbow trout	Visceral fat	Unspecified (Lowe, 1967).
	W	Spot	Liver	Cytoplasmic vacuolation (Couch, 1975).
	W	Lake trout	Gills	Hyperplasia of lamellar respiratory epithelium[1] and telangiectasia of lamellae (Walsh and Ribelin. 1975).
			Eye	Hemorrhage in anterior chamber (Walsh and Ribelin, 1975).
			Skeletal muscle	Hemorrhage near vertebrae and atrophy near lateral line (Walsh and Ribelin, 1975).
			Fat	Myxomatous degeneration (Walsh and Ribelin, 1975).
			Spleen	Hypocellularity and fibroplasia (Walsh and Ribelin, 1975).
			Liver	Fatty change (Walsh and Ribelin, 1975).
			Brain	Vacuolation of the lateral geniculate body (Walsh and Ribelin, 1975).
	W	Coho salmon	Integument	Erosion of premaxillary region[1] (Walsh and Ribelin, 1975).
			Skeletal muscle	Hemorrhage near vertebrae and atrophy near lateral line (Walsh and Ribelin, 1975).
			Fat	Myxomatous degeneration (Walsh and Ribelin, 1975).
			Spleen	Hypocellularity and fibroplasia (Walsh and Ribelin, 1975).
			Liver	Fatty change (Walsh and Ribelin, 1975).
			Brain	Vacuolation of molecular layer of optic tectum (Walsh and Ribelin, 1975).
	W	Bent-nosed clam (*Macoma nasuta*)	Gills; mantle; siphon; suprabranchial gland	Epithelial vacuolation, pyknosis and necrosis with ultimate sloughing in the first 3 tissues (Armstrong and Millemann, 1974).
Propoxur	W	Carp fry	Gills	Increased mucus secretion, respiratory epithelial hyperplasia with "clubbing" and fusion of lamellae, vascular congestion, telangiectasia and desquamation (Lakota et al., 1978).
			Liver	Hepatocyte degeneration (Lakota et al., 1978).
Miscellaneous herbicides				
Acrolein	W	Coho salmon	Gills	Respiratory epithelium totally destroyed, necrotic, and sloughed (Hendricks, 1979).
			Esophagus	Massive necrosis of all 4 tunics (Hendricks, 1979).
			Liver	Separation of hepatocytes within muralia and some necrosis (Hendricks, 1979).
			Kidney	Congestion with considerable debris and precipitate in tubule lumina, severe necrosis of both segments of proximal tubule in one case (Hendricks, 1979).
Amitrole-T	W	Coho salmon	Gills	Lamellar respiratory epithelium with hydropic degeneration showing separation from underlying pillar cells (Hendricks, 1979).
			Liver	Hepatocytes showing either hydropic degeneration or diffuse coagulative necrosis in approximately 25 percent of parenchymal cells (Hendricks, 1979).
			Kidney	Coagulative necrosis of all regions of the nephrons and most of the hematopoietic tissue (Hendricks, 1979).
Atrazine	W	Lake trout	Integument	Hypersecretion of mucus (Walsh and Ribelin, 1975).
			Gills	Hypersecretion of mucus, hyperplasia of lammelar respiratory epithelium[1], and telangiectasia[1] (Walsh and Ribelin, 1975).
			Skeletal muscle	Edema of myotomes (Walsh and Ribelin, 1975).
			Spleen	Paleness, atrophy, hypocellularity with fibroplasia (Walsh and Ribelin, 1975).
			Abdomen; stomach	Distension with thick mucus (Walsh and Ribelin, 1975).
			Stomach	Edema[1] (Walsh and Ribelin, 1975).
			Intestine	Hyperemia and edema[1] (Walsh and Ribelin, 1975).
			Pyloric cecae; ureters; urinary and swim bladders	Edema (Walsh and Ribelin, 1975).
	W	Coho salmon	Integument	Erosion of premaxillary region and hypersecretion of mucus (Walsh and Ribelin, 1975).
			Gills	Hypersecretion of mucus, hyperplasia of lamellar respiratory epithelium[1] and telangiectasia[1] (Walsh and Ribelin, 1975); hypertrophy of gill respiratory epithelium and large aneurysms at the base of filaments (Hendricks, 1979).
			Eye	Exophthalmos (Walsh and Ribelin, 1975).
			Skeletal muscle	Edema of myotomes (Walsh and Ribelin, 1975).
			Spleen	Hypocellularity[1] and fibroplasia[1] (Walsh and Ribelin, 1975).
			Stomach; abdomen	Distension with thick mucus (Walsh and Ribelin, 1975).
			Stomach	Edema[1] (Walsh and Ribelin, 1975).
			Pyloric cecae; swim and urinary bladders; ureters	Edema (Walsh and Ribelin, 1975).
			Intestine	Hyperemia and edema[1] (Walsh and Ribelin, 1975).
Dinoseb	W	Coho salmon	Gills	Total necrosis and sloughing of lamellar respiratory epithelium (Hendricks, 1979).
			Liver	Diffuse necrosis of parenchymal cells (Hendricks, 1979).
			Kidney	Necrosis of all regions of tubules and most of hematopoietic tissue (Hendricks, 1979).
Diquat	W	Coho salmon	Gills	Hypertrophy and hyperplasia of lamellar and interlamellar epithelium (Hendricks, 1979).
			Liver	Several foci of degenerate and occasionally necrotic parenchymal cells (Hendricks, 1979).
			Kidney	Numerous degenerate and some necrotic tubule cells (Hendricks, 1979).
Hydrothol 191	W	Redear sunfish (*Lepomis microlophus*)	Gills	Reversible respiratory epithelial hyperplasia with lamellar and filament fusions (Eller, 1969).
			Liver	Inflammation, pigmented swollen hepatocytes progressing to bizarre cells and distorted cords (Eller, 1969).
			Testes	Development of ova-like cells in follicles (Eller, 1969).
			Blood vessels	Spherical purple-red deposits within lumina (Eller, 1969).

Table 2.—Continued.

Contaminant	Exposure route	Species	Tissues affected	Lesions
Paraquat-CL	W	Coho salmon	Gills	Almost complete sloughing of respiratory epithelium while remaining epithelial cells were degenerate or necrotic (Hendricks, 1979).
			Liver	Low grade hydropic degeneration particularly in centrilobular areas (Hendricks, 1979).
			Kidney	Necrosis of cells in first and second proximal tubules (Hendricks, 1979).
$NaTA_2$	W	Carp	Gills	Hyperplasia of mucus cells with eventual necrosis of lamellar epithelium (Schultz, 1970).
			Skeletal muscle	Degenerative atrophy of myotomes (Schulz, 1970).
Trifluralin	W	Sheepshead minnow	Vertebral column	Vertebral dysplasia characterized by near-symmetrical hypertrophy of vertebrae with foci of osteoblasts and fibroblasts actively producing bone and bone precursors, hypertrophy of notochord, compression of spinal cord from vertebral outgrowth and fusion of vertebrae (Couch et al., 1979).
			Kidney	Compression of mesonephric ducts from outgrowth of vertebrae (Couch et al., 1979).
Other compounds				
Bis(tri-n-butyltin) oxide	W	Rainbow trout	Gills	Respiratory epithelial degeneration with separation from basement membrane, vacuolation and vasodilation; chloride cell degeneration with hypertrophied perinuclear space and smooth endoplasmic reticulum, mitochondria having disorganized cristae and ruptured membranes (Chliamovitch and Kuhn, 1977).
			Liver	Sinusoid congestion, thinning and separation of biliary epithelium from basement membrane accompanied by some necrosis (Chliamovitch and Kuhn, 1977).
			Integument	Excessive mucus secretion (Chiliamovitch and Kuhn, 1977).
			Eye	Corneal degeneration with necrosis of external squamous epithelium and vacuolation of basal epithelium; hemorrhage and leucocyte infiltration of limbus corneae (Chliamovitch and Kuhn, 1977).
DMSO	IP	Rainbow trout, chinook salmon, coho salmon, sockeye salmon (*Oncorhynchus nerka*)	Gills	Swelling, fusion and edema of lamellae with moderate respiratory epithelial hypertrophy, some necrosis and desquamation; hypertrophy of albuminous gland cells (Benville et al., 1968).
			Kidney	Glomerular edema with cellular debris in Bowman's space; hypoplasia, pyknosis and karyorrhexis of hematopoietic cells (Benville et al., 1968).
			Liver	Subcapsular and portal necrosis (Benville et al., 1968).
			Pancreas	Subcapsular necrosis (Benville et al., 1968).
			Spleen	Subcapsular necrosis, pyknosis and karyorrhexis of blood cells (Benville et al., 1968).
			Brain; meninges	Engorgement and dilation of vessels; cerebral edema (Benville et al., 1968).
SLS	W	Rainbow trout	Gills	Hypertrophy, necrosis, and desquamation of filamental and lamellar epithelium with enlarged intercellular lymphoid spaces infiltrated by lymphocytes and granulocytes; chloride cells with pyknotic nuclei; occluded vascular spaces and hematomas present (Abel and Skidmore, 1975).
	W	Goldfish	Adrenal cortex (interrenal cells)	Increase in nuclear diameter and number and size of nucleoli with more extensive and intensively staining cytoplasm; atrophy of cells with clumping of nucleoplasm (Bromage and Fuchs, 1976).
TEPA	W	Guppy	Testes	Atrophy and hypospermia (Stock and Cope, 1969).
TFM	W	Rainbow trout	Gills	Erythema of pharyngeal area, heavy mucus secretion and lamellar edema (Christie and Battle, 1963).
	W	Lamprey (*Petromyzon marinus*)	Gills	Erythema and swelling of pharyngeal area, heavy mucus secretion and lamellar edema (Christie and Battle, 1963).
			Liver	Erythema (Christie and Battle, 1963).
			Skeletal muscle	Myotomal edema (Christie and Battle, 1963).
			Cloaca	Erythema and increased mucus secretion (Christie and Battle, 1963).
Acid-alkaline water H_2SO_4-NaOH	W	Brook trout (*Salvelinus fontinalis*) Ranges of pH		
		>9.0 <5.6	Gills	Separation of endothelial and epithelial cells from pillar cells (Daye and Garside, 1976).
		>9.0 <4.8	Cornea	Desquamation of corneal epithelium often preceded by loss of distinct stratification of epithelial cells and/or poor differentiation between epithelium and substantia propria; considerable swelling of substantia propria following sloughing of epithelial layer during acute alkaline conditions (Daye and Garside, 1976).
		≥9.5 - 2.2	Eye lens	Degenerative changes consisting of disorganized structure of capsule, lens epithelium, and fibers such that boundaries become indistinct; enlargement of capsule at extreme acid pH (Daye and Garside, 1976).
		>9.5 <4.8	Olfactory organs	Hypertrophy and increase in numbers of goblet cells in olfactory epithelium; cellular vacuolation progressing to "condensation of nuclear bodies" (pyknosis) in cells of lamina propria (Daye and Garside, 1976).
		>9.0 ≤4.2	Integument	Hypertrophy and hypersecretion of mucus goblet cells; degeneration, condensation of nuclear bodies and desquamation of stratified epithelial cells of epidermis followed by degenerative changes in exposed dermis (Daye and Garside, 1976).
		>10 <2.0	Esophagus	Vacuolation and nuclear condensation of mucosal epithelial cells (Daye and Garside, 1976).
Metals and nitrogenous compounds				
Ammonia	W	Rainbow trout	Gills	Swollen filaments often protruding beyond opercula, hypertrophy and severe hyperplasia of respiratory epithelium with edema accompanied by fusion of lamellae and filaments; presence of telangiectasia in lamellar sinusoids with necrotic erythrocytes and infiltrating leukocytes (Smith and Piper, 1975).
			Liver	Cytoplasmic degeneration of hepatocytes around central veins, fatty change, dilation of sinusoids, and focal necrosis of hepatic parenchyma (Smith and Piper, 1975).
			Intestine	Mild necrosis and desquamation of mucosa (Smith and Piper, 1975).
			Spleen	Reduction of lymphoid elements (Smith and Piper, 1975).
	W	Rainbow trout fry	Gills	Hypertrophy of lamellar respiratory epithelium with some karyolysis and karyorrhexis occurring at higher dosages (Burkhalter and Kaya, 1977).

Table 2.—Continued.

Contaminant	Exposure route	Species	Tissues effected	Lesions
Ammonia (cont.)	W	Rainbow trout fry (cont.)	Yolk sac	Developmental retardation with failure to absorb the yolk sac and occurrence of blue sac disease syndrome (Burkhalter and Kaya, 1977).
Cadmium 109	W	Medaka fry (*Oryzias latipes*)	Brain	Nuclear pyknosis (Aoki, 1978).
$CdCl_2$	W	Mummichog	Gills	Focal hypertrophy of filaments and hyperplasia of lamellar and filament epithelium accompanied by high mitotic index, necrosis and desquamation of epithelium and lymphocyte infiltration (Gardner and Yevich, 1970).
			Intestine	Distension of anterior portion, epithelial hypertrophy, increased secretion of mucous with mucoid casts from anus; necrosis of mucosa with desquamation of epithelial cells; edema, focal necrosis and lymphocytic infiltration of submucosa (Gardner and Yevich, 1970).
			Peripheral blood	Numbers of eosinophilic granulocytes increased, some having deformed nuclei, cytoplasmic vacuolation, and reduction in granular mass; nuclei of thrombocytes became irregularly shaped (Gardner and Yevich, 1970); poikilocytosis and anisocytosis primarily in erythrocytes (Gardner, 1975).
			Thyroid gland	Possible hyperplasia of follicles? (Gardner, 1975).
			Kidney	Reduction of pronephric numbers of mature eosinophilic granulocytes (Gardner and Yevich, 1970).
	W	Brook trout	Testes	Purple-brown mottling throughout, necrosis of tubular boundary cells, hemorrhage, vasodilation, and congestion (Sangalang and O'Halloran, 1972).
	W	Cunner	Gills	Equivocal lesions, possibly artefactual (Newman and MacLean, 1974).
			Integument	Swelling of epithelial cells and reduced mucus secretion (Newman and MacLean, 1974).
			Peripheral blood	Poikilocytosis, karyorrhexis and abundant "smudge" cells among erythrocytes; lymphocytopenia, thrombocytopenia and neutrophilia (Newman and MacLean, 1974).
			Intestine	Swollen mucosal epithelium with hypertrophied nuclei and prominent nucleoli; necrosis and desquamation of mucosal epithelium with cellular debris and mucus in lumen (Newman and MacLean, 1974).
			Kidney	Diffuse tubular necrosis with sloughed epithelial cells and hyalin casts in dilated tubule lumina; erythrophagocytosis and reduction or absence of hemosiderin (Newman and MacLean, 1974).
	IP	Goldfish	Testes	Lower mean index of spermatogenic development; increased numbers of macrophages with phagocytized debris forming granulomas in some cases; necrosis of primary germ cells with atrophy of seminiferous tubules resulting in fibrosis and infiltration of mononuclear inflammatory cells (Tafanelli and Summerfelt, 1975).
			Ovaries	Decreased frequency of oocyte maturation (Tafanelli and Summerfelt, 1975).
			Kidney	Abundant interstitial macrophage accumulation usually forming granulomas, some tubular atrophy (Tafanelli and Summerfelt, 1975).
			Liver	Formation of macrophage granulomas (Tafanelli and Summerfelt, 1975).
	W	Sapo (*Halobatrachus didactylus*)	Peripheral blood	Erythrocyte anisocytosis, poikilocytosis, anisochromsia, microcytosis, karyorrhexis, cyroplasmic vacuolation and hyperchromasia, presence of fusiform shaped cells, and morphological alterations of red blood cell nuclei (Gutierrez et al., 1978).
			Intestine	Loss of normal nuclear orientation in mucosal epithelium, nuclear hyperchromasia, cytoplasmic vacuolation and epithelial necrosis with debris in lumen (Gutierrez et al., 1978).
			Liver	Increase in connective tissue and numbers of hepatocyte nuclei (Gutierrez et al., 1978).
			Kidney	Renal tubular degeneration, nuclei not in normal position and hyperchromatic, and dilated lumina filled with amorphous eosinophilic material (Guitterez et al., 1978).
	W	Spot	Kidney	Focal degeneration of first and second proximal tubules with granular, basophilic, and vacuolated cytoplasm containing swollen or dense contracted mitochondria, some with a granular matrix and focal electron densities; third proximal tubules with increased numbers of vacuoles, lipid droplets, autophagic vacuoles, nuclei with marginated chromatin, swollen nuclear envelopes and basal membranes contorted into myelin-like figures and cellular casts in the lumina; Bowman's space slightly swollen with cell debris (Hawkins et al., 1980).
	W	Langostino (shrimp) (*Penaeus kerathurus*)	Hepatopancreas	Digestive diverticular epithelium reduced in height, tubules dilated with enlarged circular lumina (Establier et al., 1978c).
$CdCl_2$; $CdCl_2$ & $CuCl_2$; $CdCl_2$ & $ZnCl_2$	W	Mummichog	Kidney	Degenerative changes and necrosis in epithelial cells of proximal tubules containing granular casts and nuclear debris (Gardner and Yevich, 1970; Eisler and Gardner, 1973); necrotic tubule epithelium appearing as pink staining granular material with either pyknotic nuclei or nuclear debris (Eisler and Gardner, 1973).
$CuCl_2$	W & IP	Mummichog	Liver	Focal necrosis (Gardner and LaRoche, 1973).
			Kidney	Unspecified renal damage (Gardner and LaRoche, 1973).
			Lateral line canal (cephalic extension)	Necrosis of sensory and sustentacular epithelium characterized by nuclear pyknosis, karyorrhexis, swelling and reduction of staining capacity of cell cytoplasm; reduction in numbers of goblet cells lining canal and no effect on basal cuboidal cells (Gardner and LaRoche, 1973).
			Olfactory organs	Necrosis of epithelial receptor cells, hyperplasia of sustentacular epithelium with cyst-like formations containing cellular debris and remnants of sensory tissue (Gardner and LaRoche, 1973).
	W	Atlantic silverside	Lateral line canal (cephalic extension)	Changes similar to those above in the mummichog (Gardner and LaRoche, 1973).
			Olfactory organ	Complete necrosis of all cellular elements accompanied by vasodilation and congestion of vessels in submucosa (Gardner and LaRoche, 1973).
			Brain	Vasodilation and congestion with rupture and hemorrhage of the menix primitiva (Gardner and LaRoche, 1973).
			Eye	Hemorrhaging of periorbital connective tissue (Gardner and LaRoche, 1973).
$CuCl_2$; $CuCl_2$ & $ZnCl_2$; $CuCl_2$ & $ZnCl_2$ & $CdCl_2$; $CuCl_2$ & $CdCl_2$	W	Mummichog	Kidney	Cytoplasmic vacuolation and discontinuity of cell walls in basal region of epithelium lining proximal and collecting tubules; clumping of nuclear chromatin in same cells (Eisler and Gardner, 1973).

Table 2.—Continued.

Contaminant	Exposure route	Species	Tissues affected	Lesions
$CuCl_2$; $CuCl_2$ & $ZnCl_2$; $CuCl_2$ & $ZnCl_2$ & $CdCl_2$; $CuCl_2$ & $CdCl_2$ (cont.)	W	Mummichog (cont.)	Lateral line (cephalic extension)	Pyknotic nuclei and clear cytoplasm in swollen epithelial cells beneath canal lining; occasional neuromast nuclei with enlarged nucleoli and loss of nuclei in others (Eisler and Gardner, 1973).
			Oral cavity	Focal pyknosis and hyperchromasia of squamous cell nuclei progressing to complete necrosis (Eisler and Gardner, 1973).
$CuCl_2$ & $ZnCl_2$ & $CdCl_2$	W	Mummichog	Gills	Significant deposits of mucus within interlamellar spaces due to hypersecretion by goblet cells (Eisler and Gardner, 1973).
$CuNO_3$	W	Zebra fish (fry) (*Brachydanio rerio*)	Central nervous system	Spirality (Ozoh, 1979a).
$CuSO_4$	W	Winter flounder (*Pseudopleuronectes americanus*)	Gills	Progressive degeneration of lamellar respiratory epithelium with vacuolation and reduction in height of epithelial layer, overabundance of enlarged chloride cells, fewer but swollen goblet cells, lamellar fusion with eventual desquamation and necrosis of epithelial layer. Ultrastructural changes in epithelium included formation of autophagosomes, vacuoles, myelin-like bodies and apical vesicles in the cytoplasm, reduction in thickness of apical homogeneous cytoplasm of epithelial cells, increased amounts of particulate material on epithelial cell surfaces (Baker, 1969).
			Liver	Centrilobular fatty change in hepatocytes (Baker, 1969).
			Kidney	Necrosis of hematopoietic tissue, size reduction and vacuolation of renal tubular epithelium with disintegration of apical cytoplasm, and presence of dense material in tubule lumina (Baker, 1969).
	W	Asian fish sp. (*Heteropneustes fossilis*)	Gills	Disorganized appearance of lamellae, vacuolation of respiratory epithelial cells, and reduction in goblet cell number (Bhatnagar and Shrivastava, 1975).
			Stomach	Degeneration of gastric epithelium (Bhatnagar and Shrivastava, 1975).
			Kidney	Necrosis of hematopoietic tissue, unspecified degeneration of cellular structure, and contraction of cytoplasm (Bhatnagar and Shrivastava, 1975).
Landfill leachates	W	Rainbow trout	Adrenal cortex (interrenal cells)	Hypertrophy of nuclei and nucleoli, vacuolation of cytoplasm, abundant mitotic figures and prominent sinusoids concommitant with increased plasma cortisol levels (McBride et al., 1979).
$C_4H_6O_4Pb$	IP	Zebra cichlid (Fry) (*Cichlasoma nigrofasciatum*)	Fins; tail	Erosion of epiethelium and atrophy (Ozoh, 1979b).
			Eyes	Anophthalmia and microphthalmia (Ozoh, 1979b).
			Heart	Hypotonicity with poor circulation and hemostasis (Ozoh, 1979b).
			Vertebral column	Lordoscoliosis (Ozoh, 1979b).
			Melanocytes	Abnormal melanogenesis of melanophores (Ozoh, 1979b).
$PbNO_3$	W	Asian catfish	Intestine; pyloric cecae	Reduction in height of villi, desquamation of degenerate and necrotic mucosal epithelium from the tips and crypts of villi with underlying infiltration of inflammatory cells in the lamina propria (Sastry and Gupta, 1978b).
			Liver	Disorganization of muralia, focal hepatocyte necrosis especially in centrilobular areas with portal and perilobular infiltration of inflammatory cells, slight perivascular fibrosis, dilation of interhepatocyte spaces, and diffuse deposition of lipofuscin granules within hepatocyte cytoplasm (Sastry and Gupta, 1978b).
	W	Zebra fish (embryos & fry)	Integument	Epitheliomas, tail erosion (Ozoh, 1979a).
			Yolk sac	Poor resorption (Ozoh, 1979a).
			Other	Suppression of $CuNO_3$ induced abnormalities (Ozoh, 1979a).
$HgCl_2$	W	Rainbow trout (fry, fingerlings)	Gills	Some degree of respiratory epithelial hyperplasia but no significant increase in mitotic activity; extensive in situ necrosis with karyorrhexis of respiratory epithelial nuclei (Wobeser, 1975a).
	W & IP	Hogchoker (*Trinectes maculatus*)	Kidney	Selective necrosis of second proximal tubules; earlier ultrastructural changes include distortion of microvilli, dilation of endoplasmic reticulum and nuclear envelopes, swelling of mitochondria with flocculent densities (Trump et al., 1975).
	W	Mummichog	Neurosensory organs	Necrosis of all cellular elements of lateral line canals, severe degenerative changes in olfactory organs (Gardner, 1975).
			Kidney	Epithelium of proximal tubules with cytoplasmic vacuolation, dilation of nuclear envelopes, mitochondrial swelling, and distortion of microvilli (Wassermann and Koepp, 1977).
	W	Asian catfish	Stomach	Loss of pepsinogen granules from chief cells, disintegration of goblet cells, pyknosis of gastric glandular epithelium, desquamation of gastric mucosa (Sastry and Gupta, 1978a).
			Pyloric cecae	Erosion of mucosa at tips of villi and infiltration of inflammatory cells (Sastry and Gupta, 1978a).
			Intestine	Similar changes as in pyloric cecae above, including a catarrhal enteritis involving hyperactive mucus secretion; erosion of mucosa at tips of villi (Sastry and Gupta, 1978a).
			Rectum	Erosion of mucosa at tips of villi (Sastry and Gupta, 1978a).
			Liver	Perilobular and centrilobular necrosis, depletion of hepatocyte glycogen, disarray of muralia, cirrhosis, and lipid deposition with infiltration of phagocytic inflammatory cells in vasculature and intercellular spaces (Sasatry and Gupta, 1978a).
	W	Sapo	Intestine	Edema of lamina propria of villi, hyperchromasia of nuclei, and apical cytoplasm of mucosal epithelium (Gutierrez et al., 1978).
	W	Lisa (*Mugil auratus*)	Intestine	Thinning and hyperchromasia of mucosal epithelium, disorganization of lamina propria (Establier et al., 1978a).
			Liver	Proliferation of dilated vascular elements, vacuolar degeneration of hepatocytes and disorganization of muralia (Establier et al., 1978a).
	W	Robalo (*Dicentrarchus labrax*)	Gills	Distal swelling of lamellar respiratory epithelium and desquamation (Establier et al., 1978b).
			Intestine	Nuclear depolarization and hyperchromasia of apical cytoplasm in mucosal epithelium accompanied by increased numbers of goblet cells and mucosal desquamation (Establier et al., 1978b).
			Liver	Hepatocyte vacuolation and degeneration, congestion of capillaries (Establier et al., 1978b).
			Kidney	Renal tubules with reduced lumina, vacuolized epithelium having depolarized nuclei and hypochromatic cytoplasm; retraction of glomerular tufts (Establier et al., 1978b).

Table 2.—Continued.

Contaminant	Exposure route	Species	Tissues affected	Lesions
CH_3HgCl_2	W	Rainbow trout (fry and fingerlings)	Gills	Hypertrophy and hyperplasia of secondary interlamellar epithelium with ballooning degeneration, separation of respiratory epithelium from lamellae due to edema fluid, abnormally high mitotic index with abnormal mitotic figures, and desquamation (Wobeser, 1975a).
	D & IP	Rainbow trout fingerlings	Gills	Swelling and hyperplasia of lamellar respiratory epithelium with clubbing and fusion of lamellae, respiratory epithelial necrosis and separation (Wobeser, 1975b).
			Pseudobranch	Swollen epithelium with occasional ballooning degeneration (Wobeser, 1975b).
			Posterior kidney	Slight swelling of parietal epithelium lining Bowman's capsules, dilation of renal tubules and Bowman's space, hydropic degeneration of tubular epithelium and scattered foci of tubulo-necrosis (Wobeser, 1975b).
	IP	Channel catfish	Liver	Periportal necrosis of exocrine pancreas and surrounding hepatocytes, desquamation of biliary epithelium into duct lumina, inflammatory exudate on surface of liver capsule (Kendall, 1977).
	W	Lisa	Gills	Respiratory epithelial hyperplasia and desquamation (Establier et al., 1978a).
			Stomach (tunica muscularis)	Nuclear depolarization, cytoplasmic hyperchromasia, and desquamation of mucosal epithelium (Establier et al., 1978a).
			Intestine	Degeneration of mucosal epithelium including hyperchromasia, vacuolation, and depolarization of nuclei (Establier et al., 1978a).
			Liver	Vacuolation of hepatocytes, proliferation and dilation of capillaries, disorganization of muralia (Establier et al., 1978a).
			Kidney	Focal degeneration of tubule epithelium with hyperchromatic nuclei and constricted lumina containing finely granular material; proliferative glomerulonephritis (Establier et al., 1978a).
NO_2	W	Steelhead trout (*Salmo gairdneri*)	Gills	Hypertrophy, hyperplasia, and cloudy swelling of lamellar respiratory epithelium (Wedemeyer and Yasutake, 1978).
AgCl	W	Mummichog	Lateral line (cephalic extension)	Necrosis of cuboidal cells lining canal but no effect on neuromast epithelium; infiltration of granular leukocytes in canal lumina, canal walls, and peripheral connective tissue (Gardner, 1975).
			Olfactory organ	Degeneration and necrosis of lining and sustentacular epithelium with cellular debris in lumina (Gardner, 1975).
Na_2HAsO_4	W	Green sunfish (*Lepomis cyanellus*)	Liver	Ultrastructural changes in hepatocytes include presence of intranuclear and intracytoplasmic electron dense particles, proliferation of smooth endoplasmic reticulum, enlarged but fewer myelin figures, increased size and numbers of lipofuscin granules, and abnormally enlarged mitochondria (Sorensen, 1976).
$NaAsO_2$	W	Bluegill	Gills	Lamellar telangiectasis (Gilderhaus, 1966).
			Liver	Fatty infiltration and focal necrosis (Gilderhaus, 1966).
			Ovaries	Cytoplasmic clumping and karyolysis of ova (Gilderhaus, 1966).
			Blood vessels	Endothelial separation and subendothelial myositis (Gilderhaus, 1966).
$ZnSO_4$	W	Goldfish	Adrenal cortex (interrenal cells)	Degeneration with irregular clumping of nuclear material and decrease in cell volume (Bromage and Fuchs, 1976).

[1]The significance of these observations is doubtful since similar changes appeared in control fish.

Table 3.—Summary of pathological changes in tissues of fish resulting from exposures to various chemotherapeutic compounds or drugs. For "Exposure routes," W = water, D = diet, IP = intraperitoneal injection.

Chemotherapeutic agent	Exposure route	Species	Tissues affected	Lesions
Bradophen	W	*Salmo* sp.	Gills	Destruction of respiratory epithelium and hyperplasia of mucus goblet cells (Reichenbach-Klinke, 1975).
Copper sulfate	W	Various fishes (i.e., carp, trout, gudgeon)	Kidney	Dilation of renal tubules and necrosis of hematopoietic tissues (Riechenbach-Klinke, 1975).
			Liver	Increase in hepatocyte lipid (Reichenbach-Klinke, 1975).
			Also see Table 2	
Dipterex	W	Carp	Spinal cord	Swelling of lipoid substance surrounding large nerve cells (Reichenbach-Klinke, 1975).
Emtrysidina	W	Carp	Kidney	Damage to tubular epithelium (Reichenbach-Klinke, 1975).
			Integument	Hyperplasia of mucus goblet cells in epidermis (Reichenbach-Klinke, 1975).
Formalin	W	Rainbow trout	Gills	Severe hypertrophy of lamellar respiratory epithelium sometimes accompanied by desquamation, pyknosis, karyorrhexis, edema between epithelium and blood capillaries, and hemorrhage (Smith and Piper, 1972).
			Pseudobranch	Nuclear swelling and hypertrophy of epithelial cells (Smith and Piper, 1972).
			Adrenal cortex (interrenal cells)	Hyperplasia and hypertrophy (Smith and Piper, 1972).
			Spleen	Reduction of lymphoid tissue (Smith and Piper, 1972).
			Kidney	Congestion of blood sinusoids, nuclear swelling, and hydropic degeneration of renal tubular epithelium (Smith and Piper, 1972).
			Peripheral blood	Increase in the number of immature erythrocytes and some hemolysis (Smith and Piper, 1972).
	W	Steelhead trout	Gills	Hypertrophy of respiratory epithelium characterized by cloudy swelling and hydropic degeneration with eventual necrotic changes of pyknosis and karyorrhexis at longer exposure periods; separation of respiratory epithelium from lamellar pillar cells (Wedemeyer and Yasutake, 1974).
	W	Spring chinook salmon	Gills	Similar degenerative changes as seen in steelhead trout, but less severe (Wedemeyer and Yasutake, 1974).
Hexa-ex	W	Carp	Integument	Partial destruction of epidermis (Reichenbach-Klinke, 1975).
Kanamycin	IP	Rainbow trout	Kidney	Degeneration of tubular epithelium (McBride et al., 1975).
Malachite Green	W	Various fishes, (i.e., carp, trout, gudgeon)	Integument, gills, gut	Slight inflammation of epithelial cells (Reichenbach-Klinke, 1975).
			Kidney	Pigmentous degeneration of tubular epithelium, and a decrease in tubular height with an increase in the nucleus-cytoplasm ratio (Reichenbach-Klinke, 1975).
Methylene blue	W or D (?)	Various fishes, (i.e., carp, trout, gudgeon)	Peripheral blood	Polycythemia which may be beneficial to convalescing fish (Reichenbach-Klinke, 1975).

Table 3.—Continued.

Chemotherapeutic agent	Exposure route	Species	Tissues affected	Lesions
Neguvon, Masoten	W	Various fishes (i.e., carp, trout, gudgeon)	Central nervous system	Swelling of non-staining surroundings of the nerve nuclei particularly in the brain and medulla (Reichenbach-Klinke, 1975).
Ozone	W	Rainbow trout	Gills	Severe hypertrophy and hyperplasia of respiratory epithelial cells resulting in lamellar fusion and eventual epithelial degeneration and necrosis (Wedemeyer et al., 1979).
			Peripheral blood	Polycythemia and increased numbers of immature erythrocytes (Wedemeyer et al., 1979).
Penicillin G procain, dihydrostreptomycin sulfate, oxytetracycline (mixture)	IP	Chinook salmon	Embryonal tissues	Teratomas on 0-15 percent (DeCew, 1972).
Sulfamethazine	D	Fall chinook salmon	Stomach	Gross swelling (Wood et al., 1957).
			Abdominal cavity	Edema throughout (Wood et al., 1957).
			Mesenteric vessels	Necrosis of arterial walls (Wood et al., 1957).
			Pancreas, adipose tissue of pyloric cecae	Marked edema (Wood et al., 1957).
			Liver	Minor degenerative changes in parenchymal cells (Wood et al., 1957).
			Kidney	Minor degenerative changes in renal tubules; crystalline deposits of sulfamethazine in hematopoietic tissue (Wood et al., 1957).
Sulfonamides	D (?)	Various fishes (i.e., carp, trout, gudgeon)	Kidney	Severe renal damage (Reichenbach-Klinke, 1975).
Thiabendazole	W or D (?)	Carp	Liver	Hypertrophy of hepatocytes, swelling of intercellular spaces, and vascular congestion (Reichenbach-Klinke, 1975).
			Kidney	Degeneration of hematopoiatic tissue and presence of casts within renal tubule lumina (Reichenbach-Klinke, 1975).
Tobramycin	IP	Coho salmon	Kidney	Moderate to complete necrosis of proximal tubules (Schneider et al., 1980).
Yomesan	D	Carp	Kidney	Degeneration of hematopoietic tissue and increased pigment deposits (Reichenbach-Klinke, 1975).

Table 4.—Review of mycotoxins, plant derivatives, and other chemical compounds tested for carcinogenicity in various fishes and shellfishes (see Table 5).

Mycotoxins	Plant derivatives	Other chemicals	
Aflatoxin B_1 (AFB_1)	Bracken (dry powder and methanol extract)	2-Acetylaminofluorine (2-AAF)	Diethylnitrosamine (DEN)
Aflatoxin B_1 (ammoniated)	Cyclopropenoid fatty acids (CPFA)	0-Aminoazotoluene (0-AAT)	Diethylstilbestrol (DES)
Aflatoxin B_{2a} (AFB_{2a})	Cycad nut meal	Dimethylaminoazobenzene (DAAB)	Dimethylnitrosamine (DMN)
Aflatoxin G_1 (AFG_1)	Cycasin	β-Aminopropionitrile (β-APPN)	Ethylnitrosourea (ENU)
Aflatoxin M_1 (AFM_1)	Gossypol	Aminotriazole (ATA)	Methylnitrosourea (MNU)
Aflatoxin Q_1 (AFQ_1)	Ipomeanol	Aroclor 1254	3-Methylcholanthrene (MCA)
Aflatoxicol (AFL)	Ipomeanone	Benzidine	N-methyl-N'-nitro-N-nitrosoguanidine (MNNG)
Citrinin	Methylazoxymethanol acetate (MAMA)	Benzo(α)pyrene (BAP)	N-nitrosomorpholine (NM)
Ochratoxin A & B	Pyrrolizidine (*Senecio*) alkaloids (PA's)	7-12 Dimethylbenz(α)anthracene (DMBA)	Nifurpirinol
Patulin		Carbarzone	Tannic acid
Rubratoxin A & B		Carbon tetrachloride (CCl_4)	Thioacetamide
Sterigmatocystine		Dichlorodiphenyltrichloroethane (DDT)	Thiourea
Versicolorin A		Dibutylnitrosamine (DBN)	Urethane

Table 5.—Summary of histopathological changes reported in fish and shellfish experimentally exposed to various potential carcinogens (abbreviations are defined in Table 4). For "Exposure routes," W = water, D = diet, E = water exposure of embryonating eggs, T = topical application of carcinogen, IIM = implantation in musculature, IPC = implantation in peritoneal cavity, I = injected, IP = intraperitoneal injection, and IM = intramuscular injection.

Carcinogen	Exposure route	Species	Lesions
Mycotoxins			
AFB_1	D	Guppy[1] (*Lebistes reticulatus*)	Liver tumor (Sato et al., 1973).
	D & E	Rainbow trout (*Salmo gairdneri*)	Liver tumor (Halver, 1967; Lee et al., 1968, 1971; Sinnhuber et al., 1968a,b, 1977; Wales et al., 1978; Hendricks et al., 1980a,c,f).
	W	Zebra fish (*Brachydanio rerio*)	None (Bauer et al., 1972).
AFB_1 (in ammoniated corn)	D	Rainbow trout	None: Incidence of liver tumors reduced to levels in control diet (Brekke et al., 1977).
AFB_1 (purified and ammoniated	E	Rainbow trout	Liver tumors: Incidence same as with untreated AFB_1 (Hendricks, unpubl.).
AFB_{2a}	E	Rainbow trout	None (Hendricks et al., 1980f).
AFG_1	D & E	Rainbow trout	Liver tumors (Ayres et al., 1971; Hendricks et al., 1980f).
AFM_1	D & E	Rainbow trout	Liver tumors (Hendricks et al., 1980f; Sinnhuber et al., 1974).
AFQ_1	D	Rainbow trout	Liver tumors (Hendricks et al., 1980d).
	E	Rainbow trout	None (Hendricks et al., 1980f).
AFL	D & E	Rainbow trout	Liver tumors (Hendricks et al., 1980f; Schoenhard et al., 1981).
Citrinin	E	Rainbow trout	None (Hendricks, unpubl.).
Ochratoxin A & B	IP	Rainbow trout	Liver: Nuclear swelling and nuclear and cytoplasmic lipid vacuolation of hepatic parenchyma (Doster et al., 1972). Kidney: Necrosis of hematopoietic tissue, glomeruli, and proximal tubules in addition to cast formation and lipid vacuolation in renal tubules (Doster et al., 1972).
	E	Rainbow trout	None (Hendricks, unpubl.).

Table 5.—Continued.

Carcinogen	Exposure route	Species	Lesions
Patulin	E	Rainbow trout	None (Hendricks, unpubl.).
Rubratoxin A & B	E	Rainbow trout	None (Hendricks, unpubl.).
Sterigmatocystine	E	Rainbow trout	Liver tumors (Hendricks et al., 1980e,f).
Versicolorin A	E	Rainbow trout	Liver tumors (Hendricks et al., 1980e,f).
Plant derivatives			
Bracken	D	Guppy	Intestinal hyperplasia (Matsushima et al., 1975).
CPFA	D	Rainbow trout	Liver tumors (Sinnhuber et al., 1976; Hendricks et al., 1980c). Promoter of liver tumors (Lee et al., 1968, 1971; Hendricks et al., 1980c,d; Schoenhard et al., 1981).
Cycad nut meal	D	Zebra fish	Liver tumors (Stanton, 1966).
Cycasin	W	Guppy	Acute degenerative changes in liver (Stanton, 1966).
Gossypol	D	Rainbow trout	Liver foci of fatty change with bizarre nuclei and hepatocellular regeneration, necrosis of hepatocytes around bile ducts, and inflammation of periductal tissue (Herman, 1970). Kidney: Thickened glomerular basement membrane and presence of casts and melanin in tubule lumina (Herman, 1970). Pyloric cecae: Thickening of tunica propria due to inflammation (Herman, 1970). Generalized deposition of ceroid pigment in liver, kidney, and spleen (Herman, 1970).
Ipomeanol	E	Rainbow trout	None (Hendricks, unpubl.).
Ipomeanone	E	Rainbow trout	None (Hendricks, unpubl.).
MAMA	W	Medaka (*Oryzias latipes*)	Liver tumors (Aoki and Matsudaira, 1977, 1980).
PA's	D	Rainbow trout	Liver: Megalocytosis, intense eosinophilia, nuclear aberrations, microdroplet fatty change and necrosis in hepatocytes with focal hepatocyte regeneration. Extensive fibrosis in hepatic parenchyma and veno-occlusive disease in the centrolobular and hepatic veins were also present (Hendricks et al., 1981a). Kidney: Thickened glomerular basement membranes at higher doses (Hendricks et al., 1981a).
Other chemicals			
2-AAF	D	Guppy	Liver tumors (Sato et al., 1973; Pliss and Khudoley, 1975).
	D	Rainbow trout	Liver tumors (Halver, 1967).
	D	Zebra fish	Liver tumors (Pliss and Khudoley, 1975).
O-AAT	W & D	Guppy	Adenomatous hyperplasia in liver (Kimura and Kubota, 1972) and liver tumors (Khudoley, 1972; Pliss and Khudoley, 1975).
	D	Rainbow trout	Liver tumors (Halver, 1967).
	D	Zebra fish	Liver tumors (Pliss and Khudoley, 1975).
DAAB	D	Bitterling (*Rhodeus amarus*)	Degenerative changes in liver (Ermer, 1970).
	W & D	Guppy	Bile duct hyperplasia (Kimura and Kubota, 1972) and liver tumors (Khudoley, 1972; Pliss and Khudoley, 1975).
	D	Rainbow trout	Liver tumors (Halver, 1967).
	D	Stickleback (*Gasterosteus aculetus*)	Degenerative changes in liver (Ermer, 1970).
	IIM & IPC	Stickleback	No tumors (Ermer, 1970).
	D	Zebra fish	Liver tumors (Pliss and Khudoley, 1975).
β-APPN	E	Medaka, *Anoptichthys jordane, Trichogaster tricopterus, Aequidens portalegrensis*, goldfish (mixed breed)	All species developed ecchordoma-like growths (Levy, 1962).
ATA	D	Rainbow trout	No tumors (Halver, 1967).
Aroclor 1254	D	Rainbow trout exposed to AFB_1 as embryos	No increase in incidence of liver tumors (Hendricks et al., 1980a).
	D	Rainbow trout (brood females prior to spawning)	Promotes increase in liver tumor frequency in progeny following embryo exposure to AFB_1 (Hendricks et al., 1981b).
Benzidine	D	Guppy	Severe toxic effect with focal necrosis, fatty change and hyperplasia of hepatic parenchyma (Pliss and Khudoley, 1975).
BAP	T	Bitterling	Epithelioma (Ermer, 1970).
	T	Carp (*Cyprinus carpio*)	No tumors (Ermer, 1970).
	I	Stickleback	No tumors (Ermer, 1970).
	T	Stickleback	Epithelioma (Ermer, 1970).
DMBA	IP, TM, IPC, T, D	Guppy	None (Pliss and Khudoley, 1975).
	W	*Poeciliopsis lucida P. Monacha*	Liver tumors (Schultz and Schultz, 1981).
Carbarzone	D	Rainbow trout	Liver tumors[2] (Halver, 1967).
CCl	D	Rainbow trout	Liver tumors[2] (Halver, 1967).
DDT	D	Rainbow trout	Liver tumors (Halver, 1967; Hendricks, unpubl.).
DBN	E	Rainbow trout	None (Hendricks et al., 1980f).
DEN	W	Guppy	Liver tumors (Khudoley, 1971, 1973; Pliss and Khudoley, 1975).
	W	Medaka	Liver tumors (Ishikawa et al., 1975).
	E	Rainbow trout	Liver tumors (Hendricks, unpubl.).
	W	Zebra fish	Liver tumors (Stanton, 1965; Pliss and Khudoley, 1975).
DES	D	Rainbow trout	Liver tumors[2] (Halver, 1967).
DMN	D & W	Guppy	Liver tumors (Khudoley, 1971, 1973; Sato et al., 1973; Pliss and Khudoley, 1975).
	D & E	Rainbow trout	Liver tumors (Halver, 1967; Grieco et al., 1978; Hendricks et al., 1980f; Kimura et al., 1981a).
	W	Zebra fish	Liver tumors (Pliss and Khudoley, 1975).
	W	Freshwater mussel (*Unio pictorum*)	Degeneration and focal necrosis of tubular epithelium in the digestive gland followed by epithelial hyperplasia and neoplastic transformation; multifocal accumulations of hemocytes in vesicular connective tissue of the gills, gonads, and digestive gland (Khudoley and Syrenko, 1977, 1978).
	W	Bay mussel (*Mytilus edulis*)	Congestion of visceral and branchial vasculature accompanied by a thickening of vessel walls from deposition of a collagen-like material; severe alteration of normal branchial architecture by congested vessels and hemocyte infiltration of ciliated gill epithelium; loss of frontal and laterofrontal cilia from gill epithelium; diffuse infiltration of hemocytes in vesicular connective tissue and formation of occasional discrete hemocyte granulomas with central areas of necrosis; vesiculation and atrophy of digestive tubular epithelium with tubular necrosis in areas adjacent to hemocyte infiltration (Rasmussen, 1982).
ENU	E	Rainbow trout	None (Hendricks et al., 1980f).

Table 5.—Continued.

Carcinogen	Exposure route	Species	Lesions
MNU	W	Platyfish/swordtail (*Platypoecilus variatus*/ *Xiphophorus helleri*)	Fibrosarcomas and rhabdomyosarcomas (Schwab et al., 1978).
MCA	T	Bitterling	Epithelioma (Ermer, 1970).
	T	Carp	No tumors (Ermer, 1970).
	T	Stickleback	Epithelioma (Ermer, 1970).
MNNG	E	Rainbow trout	Liver tumors, kidney tumors, air bladder adenomas (Hendricks et al., 1980b,f; Kimura et al., 1981a); inhibited maturation and tumors of the gonads, stomach adenomas, and abnormal pigmentation of the skin (Kimura et al., 1981a).
NM	W	Guppy; Zebra fish	Liver tumors and intestinal adenocarcinomas. Poorly differentiated fibrous lesion in abdominal cavities of zebra fish (Pliss and Khudoley, 1975).
Nifurpirinol	W	Croaker (*Nibea mitsukurii*)	Chromatophoromas (Kimura et al., 1981b).
Tannic acid	D	Rainbow trout	Liver tumors[2] (Halver, 1967).
Thioacetamide	D	Rainbow trout	No tumors (Halver, 1967).
Thiourea	D	Rainbow trout	Liver tumors[2] (Halver, 1967).
Urethane	D	Rainbow trout	Liver tumors[2] (Halver, 1967).

[1]A review of carcinogenesis in aquarium fish through 1975 is presented by Matsushima and Sugimura (1976).
[2]Doubtful significance, particularly in examples where massive concentrations of a compound have been used, since tumor frequency is low (5-29 percent) and not dose responsive.

(Continued from page 2.)

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Quantification of National Marine Fisheries Service Habitat Conservation Efforts in the Southeast Region of the United States

WILLIAM N. LINDALL, Jr., and GORDON W. THAYER

Introduction

"All I want to do is dredge and fill one or two acres of marsh and shallow water for my housing project. Surely this small amount can't hurt anything when there are 15 million acres out there."
—*Land developer.*

"We recognize that the dredging and spoiling associated with our proposed access canal will destroy eight acres of marshland, but the Nation needs the oil and there are millions of acres of marshland out there."
—*Oil company representative.*

These are typical statements heard almost daily by National Marine Fisheries Service (NMFS) biologists when permit applicants learn from the U.S. Army Corps of Engineers that NMFS has recommended that their proposed projects be modified to reduce adverse effects on fishery habitat, or denied altogether.

Granted, most small development projects, when considered individually, would not substantially affect fishery resources. However, the collective loss associated with numerous projects raises valid questions that resource managers must answer: How much of each habitat type is proposed for destruction or alteration each year? Where is it located? How much habitat is actually permitted to be lost? What are the cumulative effects of these habitat alterations on fishery resources?

The NMFS Environmental Assessment Branch (EAB) is collecting data to help answer these questions as they relate to to NMFS involvement in the Corps of Engineers permitting program. In this paper we summarize data collected during the first year (October 1980-September 1981) that NMFS has quantified its recommendations. The need for these data is explained and the interaction between the NMFS Southeast Fisheries Center (SEFC) and Southeast Regional Office (SERO) is discussed.

ABSTRACT — Fiscal year 1981 (October 1980-September 1981) was the first year the National Marine Fisheries Service quantified the cumulative acreage of habitat involved in the Corps of Engineers permit program in the Southeast Region of the United States. NMFS made recommendations on 1,380 permit applications involving 17,969 acres. Of that total, 18 percent was proposed for dredging, 36 percent for filling, and 46 percent for impounding. NMFS did not object to alteration of 4,598 acres, recommended against altering 13,371 acres, and recommended that 3,324 acres either be restored or modified from upland habitat to mitigate the losses that were permitted. NMFS recommendations were incorporated into permits in 98 percent of the cases. Compliance with permit conditions ranged from 100 percent in the Charleston and Savannah Districts to 36 percent in the Mobile District. Because NMFS recommendations are based heavily on state-of-the-art information provided by its research laboratories, the relationship between the NMFS Southeast Regional Office and Southeast Fisheries Center laboratories is discussed briefly.

Background

The NMFS Southeast Regional Office and Southeast Fisheries Center are responsible for the protection, management, and development of marine fisheries and their habitat from North Carolina to Texas and Puerto Rico and the U.S. Virgin Islands. This area, which includes both the territorial seas and the Fishery Conservation Zone out to 200 miles (Fig. 1), contains about 29,900 miles of tidal shoreline (Shalowitz, 1964) and 17.2 million acres of marsh/estuarine habitat. The more than 300 estuarine systems represent about 60 percent of the total remaining estuarine habitats in the contiguous United States, and 46 percent of the total including Alaska.

The immense importance of estuarine-marine habitat to commercial and recreational marine fisheries in the Southeast is well documented (Smith, et al., 1966; Lauff, 1967; Jackson, 1969; Douglas and Stroud, 1971; Chabreck, 1973; Thayer et al., 1975; Turner, 1977; Peters et al., 1979; Hoss and Hettler, 1981; Thayer and Ustach, 1981). The majority of species important to commercial and recreational

William N. Lindall, Jr., is with the Environmental Assessment Branch, Southeast Regional Office, National Marine Fisheries Service, NOAA, 9450 Koger Boulevard, St. Petersburg, FL 33702. Gordon W. Thayer is with the Beaufort Laboratory, Southeast Fisheries Center, NMFS, NOAA, Beaufort, NC 28516. This article is contribution No. 82-24B of the NMFS Southeast Fisheries Center.

fisheries reproduce, grow, and are ultimately harvested in estuaries.

The complex of fisheries habitat, which extends from the freshwater-saltwater interface to the oceanic pelagic and benthic habitats (including inshore and offshore reefs and rocky outcroppings which support a valuable reef-fish fishery), is being threatened by many human activities. These include municipal and industrial waste disposal, water diversion through damming and channelizing, dredging and filling, real estate development, marine transportation, and mineral and energy exploration and production. Projected industrial development and population growth for the Southeast Region dictate that continued adverse effects on fisheries habitats be minimized or reversed if fisheries production is to be maintained or increased.

NMFS efforts toward conservation of estuarine habitat represent the cornerstone of marine fishery management in the Southeast Region, since about 96 percent of commercial and over 50 percent of recreational fishery landings in the Southeast Region consist of fish and shellfish that use the estuary for part or all of their lives. The commercial yield of these species in the Southeast Region was about 43 percent of U.S. landings for all fisheries for 1978-80, and included the top poundage fishery (menhaden) and the most economically valuable fishery (shrimp). The marine recreational yield constituted about 58 percent of the total recreational landings in the United States.[1]

In a typical year, the U.S. Army Corps of Engineers sends about 6,000 permit applications to the NMFS Southeast Regional Office for review. This does not include large projects authorized by Congress, such as ship and barge channels, dams, and hurricane levees. By law, NMFS is required to assess the potential impacts each project may have on fishery resources and to recommend whether the project be approved, denied, or modified. The validity of this assessment process is directly related to availability of scientific information to document the effect of habitat alterations on the productivity.

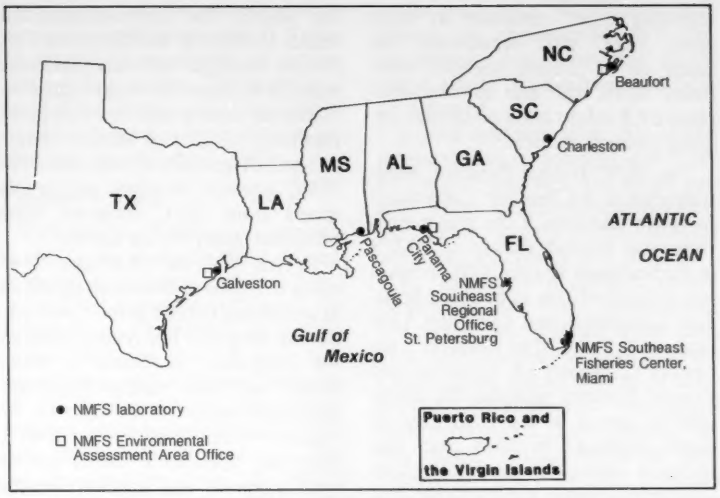

Figure 1.—Location of NMFS Laboratories, Environmental Assessment Offices, and Regional and Center headquarters in the Southeast Region.

The EAB, having no independent research capacity, relies on SEFC laboratories and state fishery agencies for information. Since available information is incomplete or imperfect, it is impossible to judge the quantitative importance of fishery habitats and to predict the potential loss to fishery production caused by alteration, loss, or degradation of habitat. Quantitative estimates of such losses cannot be made with assurance until the limits of fishery production in unaltered habitats are known.

To fulfill its environmental assessment responsibility, SERO has 14 fishery biologists strategically located in four offices (Fig. 1). Four biologists in the Galveston, Tex., office cover coastal Texas and Louisiana; five in the Panama City, Fla., office cover coastal Mississippi, Alabama, Florida, Puerto Rico, and the Virgin Islands; three in the Beaufort, N.C., office cover coastal North Carolina, South Carolina, and Georgia; and two in the Regional Office, St. Petersburg, Fla., are responsible for overall program management. Since this staff is not large enough to conduct all necessary site reviews of both the permitted and congressionally authorized projects, NMFS relies on private contractors to provide site-specific information on which to formulate recommendations.

Because the EAB's recommendations on permit applications must be based on state-of-the-art research data to be valid and defensible, there is close cooperation between EAB staff and estuarine research personnel in the SEFC. The EAB not only uses SEFC publications for data but also consults directly with researchers as the need arises. One example of this cooperation is the joint development of guidelines and criteria used by the EAB in evaluating proposed projects. Another is the technical advising and counseling

[1]Southeast Fisheries Center. 1980. AECOS, alterations of estuarine-coastal-oceanic systems, a comprehensive regional program of ecological research, monitoring and information synthesis to provide decision-makers with the scientific information needed to conserve habitats of living marine resources. Report on file at Southeast Fisheries Center, NMFS, NOAA, Miami, Fla., 64 p.

of EAB biologists by SEFC staff regarding expert testimony in court cases; SEFC staff occasionally are called on to provide scientific testimony. SEFC staff also have incorporated EAB information needs into ongoing research projects.

The SEFC Habitat Program, headquartered at the Beaufort Laboratory in North Carolina, is the major environmental research element of NMFS in the Southeast Region. This program has research efforts at both the Beaufort Laboratory and Galveston Laboratory in Texas. The Beaufort Laboratory's Division of Estuarine and Coastal Ecology provides the major NMFS support to the EAB through expert testimony in court cases and technical advice in scientific matters concerning coastal and estuarine processes. The Division conducts several closely related programs concerned with aspects of fisheries ecology in the Southeast Region and with effects of contaminants (primarily heavy metals) and habitat loss on ecologically and economically important species.

Division personnel are working under contract with the Coastal Engineering Research Center of the U.S. Army Corps of Engineers, Fort Belvoir, Va., to evaluate the potential of using submerged seagrasses to stabilize subtidal disposal areas. This effort, concerned with mitigation and enhancement, is to our knowledge the only such research now conducted by the NMFS. The Environmental Research Division at the Galveston Laboratory manages contracts concerned with the influence of energy-related activities on shrimp and reef fish in the northwestern Gulf of Mexico. Details of research activities of these and other SEFC research programs can be obtained from SEFC Program Plans published yearly by the SEFC.

In early 1980, the EAB began developing a system to document its efforts in conserving habitat in the Corps permitting program. The system, designed for computer processing, is based largely on a wide variety of information from reports of contractors, including: Permit application numbers; date and applicant's name; project type, purpose, and location; habitat type (e.g., marsh species, bottom type); area (acres) proposed to be dredged, filled, or impounded; area NMFS recommended be conserved or did not object to its being altered; and NMFS recommended mitigation or compensation. The results of the first year's attempt at quantification are summarized in the next section.

Results

For the period 1 October 1980-30 September, 1981, the EAB received 6,399 permit applications from the Corps of Engineers for review and comment. Of these, 4,651 (73 percent) were given a "no objection" response because impacts were determined to be minimal, based on guidelines and criteria developed by EAB and SEFC personnel (available from the St. Petersburg office), and 1,380 (22 percent) were assigned to contractors to gather on-site information. The remaining 368 (5 percent) were not assessed because of insufficient manpower to process them, or because the public notice indicated that the Corps of Engineers would not consider recommendations other than those concerning navigation and national security.

Table 1 summarizes NMFS habitat conservation efforts, by state, in terms of the number of permit applications assigned to contractors for field review (column 1) and the acreage of habitat involved in dredging, filling, impounding, and mitigating. Region-wide quantification of each of these categories is described briefly below.

Dredging

Nearly 3,200 acres were proposed for dredging during fiscal year 1981 (Table 1, column 2). Over 80 percent of this acreage was in three states: Louisiana (38 percent), Texas (25 percent), and Florida (18 percent). NMFS did not object to dredging 1,846 acres (column 5), but recommended against dredging 1,351 acres (column 8), the

Table 1.—Number of proposed projects and acres of habitat involved in NMFS habitat conservation efforts from October 1980 to September 1981, by state. Numbers in parentheses refer to columns discussed in text.

State	No. of permit applications (1)	Acreage proposed by applicants			Acreage NMFS allowed or did not object to			Potential acreage conserved			Mitigation recommended by NMFS	
		Dredge (2)	Fill (3)	Impound. (4)	Dredge (5)	Fill (6)	Impound. (7)	Dredge (8)	Fill (9)	Impound. (10)	Restore acreage (11)	Generate acreage (12)
Louisiana	214	1,218.4	2,786.3	5,637.9	824.0	1,590.8	0	394.4	1,195.5	5,637.9	422.9	713.0
Texas	136	798.7	971.0	1,154.6	684.1	576.3	150.5	114.6	394.7	1,004.1	1,858.9	25.5
Mississippi	53	71.9	191.7	<0.1	53.6	0.7	0	18.3	191.0	<0.1	4.0	17.0
Alabama	74	81.1	1,117.6	50.0	47.4	3.3	0	33.7	1,114.3	50.0	30.4	4.3
Florida	598	588.0	1,008.6	0.7	143.9	261.7	0	444.1	746.9	0.7	87.0	47.4
Georgia	39	181.4	30.1	0	31.1	4.9	—	150.3	25.2	—	3.1	2.7
South Carolina	134	107.4	105.4	1,451.5	30.3	8.7	0	77.1	96.7	1,451.5	4.8	9.8
North Carolina	115	130.5	45.0	41.7	25.3	16.4	26.7	105.2	28.6	15.0	42.7	0
Puerto Rico	17	20.1	179.8	0	6.5	111.5	—	13.6	68.3	—	50.9	0
Virgin Islands	0	—	—	—	—	—	—	—	—	—	—	—
Year total	1,380	3,197.5	6,435.5	8,336.4	1,846.2	2,574.3	177.2	1,351.3	3 861.2	8,159.2	2,504.7	819.7

majority of which were in Florida (33 percent) and Louisiana (29 percent).

Filling

Filling was proposed on 6,435 acres (column 3): 43 percent in Louisiana, 18 percent in Alabama, 16 percent in Florida, and 15 percent in Texas. The NMFS did not object to fill on 2,574 acres (column 9). Thus, the NMFS recommended against permit issuance on 60 percent of the acreage proposed, nearly all of which was in Louisiana (31 percent), Alabama (29 percent), Florida (19 percent), and Texas (10 percent).

Impounding

Regionwide, 8,336 acres were proposed for impounding (column 4). Except for Georgia, Puerto Rico, and the Virgin Islands, impoundments were proposed in every state and ranged from less than 0.1 acre in Mississippi to more than 5,600 acres (about 9 square miles) in Louisiana. The NMFS recommended against all but 177 acres (2 percent of total proposed), all of which were in Texas and North Carolina (column 7). The total area protected from potential impoundment was 8,159 acres.

Mitigation

To offset habitat altered or destroyed by project construction (columns 5, 6, and 7), the NMFS recommended that 2,504 acres be restored and 819 acres modified to productive estuarine habitat from nonestuarine habitat (columns 11 and 12). Seventy-four percent of the acreage recommended for restoration was in Texas; 87 percent recommended for generation was in Louisiana. An example of this habitat generation is the creation of an intertidal area by grading an upland area (Lindall et al., 1979).

Cumulative Totals

Collectively, 17,969 acres were proposed to be dredged, filled, or impounded in the region (columns 2 + 3 + 4). Over one-half (9,642 acres) was in Louisiana. The NMFS did not object to alteration of 4,598 acres (columns 5 + 6 + 7), or about 26 percent of the total proposed, but recommended against alteration of 13,371 acres (columns 8 + 9 + 10). To offset permitted habitat losses, the NMFS recommended restoration and modification of 3,324 acres (columns 11 + 12). Thus, NMFS efforts conserved 16,695 acres of habitat over the year through a combination of recommending against unnecessary alteration and recommending mitigation associated with permitted alterations (i.e., those determined by the Corps to be in the public interest).

Impact of NMFS Recommendations

Our data document for the first time the magnitude of coastal fishery habitat involved in the Federal permitting process in the southeastern region of the United States and demonstrate that substantial quantities of habitat that sustain the nation's fishery resources can be protected if NMFS recommendations are implemented. However, the summary data do not show the extent to which NMFS recommendations are followed.

Logical questions arise: Are NMFS recommendations accepted and, if so, are they incorporated into issued permits? When the recommendations are incorporated into the permits, does the permit holder comply with them?

To determine if NMFS recommendations had been incorporated, 110 permits issued in the region since 1978 were randomly selected from the Corps' seven district offices to compare the issued permits with NMFS recommendations. Each project site was visited, and the results are summarized in Table 2. Of the 110 permits reviewed in the follow-up, NMFS recommendations had been incorporated into 108 (over 98 percent). Of the 80 projects completed or underway (30 had not yet begun), at least 58 (73 percent) complied with permit conditions and 14 (18 percent) did not. Corps enforcement personnel, notified of the violations, indicated they would take appropriate action. The remaining 8 (entered in Table 2 as unknown) are permits for oil and gas development in Louisiana and have been conditioned to require restoration upon abandonment of the well site (i.e., remove spoil from the marsh and return it to the canal). In these cases, compliance cannot be determined in advance.

Our basic data represent a conservative measure of our success in protecting the fishery habitat. Knowing that the NMFS position is one of strong opposition to unnecessary alteration, an increasing number of potential permit applicants consult with NMFS (either through personal meetings or through written requests for guidelines and criteria) prior to solidifying their plans and applying for a Corps of Engineers permit. Although the amount of habitat conserved through this pre-application process cannot be measured, it is undoubtedly large.

Another area of habitat conservation not reflected in our data is NMFS

Table 2.—Summary of Corps of Engineers acceptance of NMFS recommendations and applicants' compliance with permit conditions (by Corps District).

Corps District	No. of permits reviewed	NMFS recommendations incorporated in permit		Project Status		Applicants complied with permit conditions	
		Yes	No	Completed or underway	Not yet begun	Yes	No
Galveston	14	14		8	6	7	1
New Orleans	19	19		17	2	9	Unknown
Mobile	18	17	[1]1	14	4	5	9
Jacksonville	15	14	[2]1	12	3	11	1
Savannah	5	5		3	2	3	0
Charleston	15	15		7	8	7	0
Wilmington	24	24		19	5	16	3
Total	110	108	2	80	30	58	14

[1]Wording in permit was vague, thus allowing applicant to exceed boundaries of NMFS recommendation.
[2]NMFS notified, but chose not to appeal because of mitigation measures performed by the applicant.

involvement in the review of Congressionally-authorized Federal projects, which often extend over several years from initial planning to project completion. Again, the amount of habitat conserved is difficult to measure accurately, but the acreage that can be conserved or lost is potentially large. For example, the NMFS is presently attempting to convince the Corps of Engineers to prohibit filling of nearly 2,300 acres of Nueces Bay, Tex., with spoil from the Corpus Christi Ship Channel. The habitat that could be lost in this single Federal project is more than twice that in all of the proposed permitted projects on the Texas coast from October 1980 through September 1981.

Conclusions

The NMFS has been very effective in getting its conservation recommendations included in Corps of Engineers permits, but only partially effective in achieving compliance. About one in five projects investigated were in violation of permit conditions. Among the Corps of Engineers' Districts, the percentage of violations ranged from zero in the Charleston and Savannah Districts to 64 percent (9 of 14) in the Mobile District. Violations ranged from minor discrepancies to total disregard of permit conditions (i.e., constructing the project as originally proposed, despite permit conditions). The NMFS is working with the Corps of Engineers to resolve the problem of permit violations and will continue to follow-up on at least 100 projects each year.

The cumulative acreage associated with numerous small projects is considerable, and more information is needed on the kinds of habitat involved so that trends in alteration by habitat type can be tracked. The NMFS Southeast Regional Office is presently adding this capability to the present data base and associated computer program.

Finally, the quantitative relationship between habitat and fishery production needs to be known so that the changes in fish and shellfish production resulting from habitat alteration can be accurately predicted. Research at SEFC laboratories is helping to provide this information. One commonly cited estimate of the annual value of an estuary to commercial and recreational fisheries is $100/acre, which is equal to $2,000/acre capitalized at 5 percent (Gosselink, et al., 1974). Thus, the habitat preserved as a result of NMFS recommendation is worth, on the basis of these figures, over $33 million (16,695 acres × $2,000) to the commercial and recreational fishing industries of the southeastern United States.

Acknowledgments

We are grateful for the assistance of the following EAB staff in providing the basic data for this paper: Randall Cheek, Larry Hardy, Ron Sechler, and Arlene Bossie in Beaufort, N.C.; Edwin Keppner, Mark Thompson, Gary Nelson, David Nixon, Sarah Dewberry, and Deborah Talley in Panama City, Fla.; Donald Moore, Rick Ruebsamen, Peggy Keney, and Jim Lyon in Galveston, Tex.; and Bonnie Stewart in St. Petersburg, Fla. The following provided critical review and constructive comments: Richard Hoogland, Andreas Mager, Richard Raulerson, Ford Cross, David Peters, Charles Manooch, and Donald Ekberg.

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Outstanding NMFS Authors Are Honored

The outstanding papers authored by National Marine Fisheries Service scientists and published in the *Fishery Bulletin* and the *Marine Fisheries Review* in 1979 and 1980 have been announced by the NMFS Publications Advisory Committee.

For the *Fishery Bulletin* in 1979, J. Roe Hunter and Stephen R. Goldberg were cited for their outstanding paper entitled "Spawning Incidence and Batch Fecundity in Northern Anchovy, *Engraulis mordax*," 77(3):641-652. Hunter is with the NMFS La Jolla Laboratory, Southwest Fisheries Center, La Jolla, Calif., and Goldberg is with the Department of Biology, Whittier College, Whittier, Calif.

Selected as the best paper in 1979 in the *Marine Fisheries Review* was "Management for Increasing Clam Abundance," 41(10):10-22. It was written by Clyde L. MacKenzie, Jr., of the NMFS Sandy Hook Laboratory, Northeast Fisheries Center, Sandy Hook, N.J.

Chosen as the best paper in 1980 in the *Fishery Bulletin* was Peter B. Adams' "Life History Patterns in Marine Fishes and the Consequences for Fisheries Management," 78(1):1-12. Adams is with the NMFS Tiburon Laboratory, Southwest Fisheries Center, Tiburon, Calif.

And, chosen as the outstanding paper in the *Marine Fisheries Review* in 1980 was "Historical Shore-Based Catch of Bowhead Whales in the Bering, Chukchi, and Beaufort Seas," 42(9-10):5-19, by Willman M. Marquette and John R. Bockstoce. Marquette is with the National Marine Mammal Laboratory, Northwest and Alaska Fisheries Center, Seattle, Wash., and Bockstoce is Curator of Ethnology at the New Bedford Whaling Museum in New Bedford, Mass. The paper was part of a special issue, "The Bowhead Whale: Whaling and Biological Research," edited by Howard W. Braham, Willman M. Marquette, Teresa W. Bray, and J. Stephen Leatherwood.

Developed in 1975, the annual outstanding publication awards program recognizes NMFS employees who have made exceptional contributions to the knowledge and understanding of the resources, processes, and organisms studied as a part of the NMFS mission.

Fishery Bulletin papers must document outstanding scientific work while *Marine Fisheries Review* papers must be effective and interpretive contributions to the understanding and knowledge of NMFS mission-related studies.

Any NMFS employee may recommend publications of the appropriate calendar year for award consideration. Authors must have been employed by the NMFS at the time the paper was published. Nominations must include the author's name, paper title and number of pages, series name and volume number, justification to support the nomination, and the name and office affiliation of the nominator.

Japanese, NMFS Scientists Study Tuna Eggs, Larvae

Work has started on a cooperative study of skipjack tuna spawning and rearing by scientists from the NMFS Southeast Fisheries Center's Honolulu Laboratory and the Kinki University Fisheries Laboratory of Wakayama, Japan, reports Richard S. Shomura, Honolulu Laboratory Director. The Japanese scientists, Teruo Harada and his assistant, Shigeru Miyashita, are world renowned experts on the spawning and aquaculture of marine fish species. They will be working with Honolulu Laboratory scientists who work under Richard Brill, leader of the Laboratory's Experimental Ecology of Tunas project. Brill's team of researchers includes Thomas K. Kazama, Fishery Biologist; Sharon D. Hendrix, a graduate student at the University of Hawaii; and Research Assistant Linda A. Barroclough.

Harada and Miyashita arrived in Honolulu on 11 June to observe the skipjack tuna spawning operations at the Kewalo Research Facility and also to make preparations for shipping samples of viable fertilized tuna eggs and live larval tunas to Japan. The purpose of shipping these live specimens to Japan is to compare the results of rearing experiments at the Kewalo Research Facility with those at the Kinki University facilities. Earlier experiments at Kewalo showed that skipjack tuna larvae failed to live beyond about 12 days and the Honolulu Laboratory scientists are trying to determine the causes of these results.

Harada returned to Japan on 20 June to begin the rearing experiments while Miyashita remained in Honolulu to help in making shipping arrangements. Miyashita returned to Japan on 3 July.

According to Kazama, by early July five shipments of viable fertilized eggs and live skipjack tuna larvae have been made to Kinki University. Although the fate of all the specimens shipped to Japan was not known, about 70 percent of the larvae survived the rigors of shipping. However, all the larvae in the first few shipments died soon thereafter. Harada and Miyashita have made plans to return during next year's skipjack tuna spawning season, said Shomura.

Salt and Seafood: A Growing Issue

Sodium Labeling Is "Key Issue" for Pacific Fisheries Technologists

The 1982 Pacific Fisheries Technologists' Annual Meeting was held at Sacramento, Calif., 21-24 March. Approximately 135 conferees attended the meeting and 40 papers were presented in six different sessions: Fishery Development, Seafood Handling and Preservation; Decomposition Indices; Energy Conservation; Salt in Seafood Products; By-product Technology; and Aquaculture.

John Spinelli, Director of the Utilization Research Division of the NMFS Northwest and Alaska Fisheries Center, served as panel moderator for the session on "Salt in Seafood Processing." Eight papers were presented, and it was believed that sodium labeling would probably emerge as the key issue facing the industry in 1983 and beyond. Fred Hill of the University of California at Davis pointed out that proposed legislation requiring sodium labeling was undoubtedly prompted by the fact that about 60 million Americans suffer from some form of or level of hypertension, and while a high intake of salt can aggravate this condition, in only 1 percent of the cases can salt be pinpointed as the cause for hypertension.

From a technological point of view, several industry members believe it would be difficult to comply with the proposed regulation that a product shall be deemed to be misbranded if it contains 120 percent more sodium than the declared mean sodium content.

In response to the sodium problem, it was felt that primary research efforts should be directed at lowering the salt in seafood products and developing processing methods and control to assure a uniform sodium content in the product.

John Spinelli

Sodium Content of Rockfish Held in RSW

Due to the holding and processing of some fish, sodium uptake is unavoidable and may fall under proposed regulations (see article in column 1). Under these proposed regulations, two major central problems are apparent: 1) The amount of sodium taken up by the tissue, and 2) the variability of the sodium in the tissue from fish to fish in the same lot. To assess these problems, salt uptake studies in refrigerated seawater (RSW) at 0° C were undertaken by the Northwest and Alaska Fisheries Center's Utilization Research Division.

In our initial studies on variability, we measured sodium uptake in yellowtail rockfish, *Sebastes flavidus*. This variation around the mean can be expressed as the standard deviation (SD) or the percent standard deviation (%SD = 100 (SD/mean)).

Samples of fish were removed from the RSW at regular intervals (0, 1, 2, 3, 4, and 7 days) and filleted. Sodium was measured in the fillets after suitable processing. Prior to being placed in RSW, rockfish fillets contain 56 mg% sodium, but after 1 day the value doubled (108 mg%). At the end of 7 days, the fillets contained 311 mg% sodium, approximately a sevenfold increase. The variability (%SD) of the sodium on a day-to-day basis was reasonably good. The initial value (56 mg%) had a %SD of 5.3% and on day 7 the variation was 17.8%. However, the day 4 sampling had a wide range of sodium values (113 to 371 mg%, %SD = 36.5%).

We feel that foaming may have been the cause of this variation since, on day 3, we removed a large quantity of stiff, heavy foam, and then again on day 4 more foam was removed. After foam removal, it was discovered that the top layer of fish, which was removed for sampling, was not covered with RSW. Therefore the sampled fish may have had erratic contact with RSW causing the wide range of uptake.

If all the sampled data on flesh sodium (day 0 to day 7) is pooled to mimic 1 week's catch, the batch average for sodium is 177 mg% with a SD of 95. The percent standard deviation is then 53.6%. The highest sodium content of all the samples was 371 mg%, falling within 120% of the overall mean of the pooled data. This preliminary experiment indicated that variation from sample to sample on a given day is relatively small (%SD of about 20%). Therefore, the controlling factor of sodium variation in the pooled data is the length of exposure to RSW, not daily variation in sodium uptake.

Jack Wekell

High Pressure Injection Used to Add Salt to Sablefish Fillets

Recently completed experiments by the Utilization Research Division (URD) of the NMFS Northwest and Alaska Fisheries Center have demonstrated that salt and other aqueous additives, such as condensed phosphates, antioxidants, and enzyme inhibitors, can be uniformly dispersed in fish fillets by high pressure injection.

Using a pilot plant injection machine developed by the URD and built by Oceanic Associates[1], Bellevue, Wash., uniform concentrations of salt varying from 2.0-5.0 percent (in water phase) were injected into sablefish fillets. The salt concentration in the fillets was easily varied by either changing the concentration of the brine and/or changing the pressure and timing of the injection pulse. Inspection of the fillets after smoking revealed no evidence of physical damage to the fillets.

The edibility characteristic of the injected fillets was indistinguishable from conventionally brined fillets. Other tests are in progress to determine whether the quality of frozen fillets can be maintained via the injection of aqueous additives such as phosphates, enzyme inhibitors, and antioxidants.

John Spinelli

[1]Mention of trade names or commercial firms does not imply endorsement by the National Marine Fisheries Service, NOAA.

Somali Fishing Industry Has Potential for Growth

Table 1.—Somali fish catch, 1971-80.

Year	Catch (× 1,000 t)	Year	Catch (× 1,000 t)
1971	[1]7.6	1976	8.3
1972	[1]7.6	1977	14.8
1973	[1]7.6	1978	8.4
1974	7.7	1979	11.0
1975	8.0	1980	[1]11.0

[1]FAO estimate.
Source: FAO "Yearbook of Fishery Statistics," 1980.

Somalia is one of the least developed countries in Africa. It has also suffered from food shortages caused by a severe drought during the early 1970's. The Somali Ministry of Fisheries and the Coastal Development Agency (CDA) want to increase the fisheries catch to help alleviate the country's food shortage.

Somali fisheries are still largely undeveloped and play a minor role in the nation's economy. However, they could become important. Somali fishermen caught only about 11,000 metric tons (t) of fish in 1980 (Table 1), a small catch for a country with over 3,000 km of coastline. Most of the Somali catch is harvested near the northeastern coast, between Alula and Bella (see map).

The Somali fishing fleet is largely artisanal. Cooperative fishermen have about 700 small boats (6 to 8 m long), and private fishermen operate an additional 100 boats of similar size. The Somali Government helped the fishermen buy and motorize these vessels under the country's 1974-78 development program. The only commercial vessels are operated by the state-owned company Somalfish, which has 11 trawlers. These vessels are based in Kismayu and are about 23-27 m long. According to Ministry sources, none of these trawlers are currently operational, due to technical problems.

The Government's recent development program has emphasized the formation of agricultural and fishery cooperatives for artisanal fishermen, as well as resettlement centers for displaced nomads. The Ministry and the CDA have organized 19 fishing cooperatives. Fishermen from these cooperatives harvest most of the country's catch, almost all of which is marine since Somalia lacks freshwater lakes and rivers.

Somalia fishermen are required to sell their catch through the cooperatives at prices set by cooperative committees. The prices vary from US$0.24-US$0.80/kg. Cold stores have been built in Kismayu, Lula, Berbera, and Las Koreh. The Government is constructing additional cold stores in Mogadishu with Japanese assistance, and in El Hamid with German (FRG) assistance. A cold store may also be built in Zeila. The fish catch is primarily marketed domestically as the Ministry has made little effort to export. The only exports are small shipments of lobster to Europe (primarily to Italy) and shark fin to the Far East.

The Somali Government allows some foreign fishing either through joint venture agreements or by licensing foreign fishermen. There are currently two joint venture companies. The first is an Italian-Somali venture

Note: Unless otherwise credited, material in this section is from either the Foreign Fishery Information Releases (FFIR) compiled by Sunee C. Sonu, Foreign Reporting Branch, Fishery Development Division, Southwest Region, National Marine Fisheries Service, NOAA, Terminal Island, CA 90731, or the International Fishery Releases (IFR), Language Services Biweekly (LSB) reports, or Language Services News Briefs (LSNB) produced by the Office of International Fisheries Affairs, National Marine Fisheries Service, NOAA, Washington, DC 20235.

called Somital, which operates three freezer trawlers. The second is an Iraqi-Somali venture called Siadco, which operates four trawlers. The foreign partners in both joint ventures provide the vessels and the Somali Government permits them to fish in Somali-claimed waters. The catch is marketed almost entirely in the respective foreign countries.

A third joint venture, formed with the U.S.S.R. in 1974, allowed the Soviets to operate 10 trawlers. This joint venture was terminated in 1977, however, when Somalia and the U.S.S.R. broke diplomatic relations over Soviet support for Ethiopia in the Ogaden border dispute. In addition to the two current joint ventures, five foreign vessels are licensed to fish in the 200-mile territorial waters claimed by Somalia. There are no details available on the licensing arrangements.

Six foreign countries and international organizations are currently providing fisheries aid to Somalia. Foreign aid to Somali fisheries exceeds US$22 million. Foreign fishery projects include:

1) The Danish International Development Agency has agreed to provide US$1.4 million for the development of Somalia's north coast fisheries.

2) Germany (FRG) is providing US$12.2 million in assistance, not including its cold store project in El Hamid.

3) The Japanese Government is funding a US$2.2 million program for the development of cold stores and fish markets.

4) The Swedish International Development Agency (SIDA) has provided US$2.0 million for a shipyard in the Mogadishu area.

5) The United Nations Development Project (UNDP) and the Food and Agriculture Organization have an ongoing fisheries development project in the Kismayu area. The UNDP also has a US$4.0 million fisheries project along Somalia's northeast coast.

6) The United Kingdom, in cooperation with SIDA and the UNDP, is providing technical assistance for the maintenance of marine engines.

Many observers believe that the Somali fishing industry has considerable potential, for Somali-claimed waters reportedly contain important stocks of sardines that are currently unutilized. The Government, however, has not assigned a high priority to the fisheries sector and has not yet implemented a coordinated fisheries development program.

The Somali catch has grown somewhat in recent years, but its growth was seriously interrupted when the Soviets terminated their assistance in 1977 (Table 1). The traditionally low status of fishing in Somalia impedes fisheries development. Few Somalis have ever fished and most consumers prefer other types of meat. The fishery cooperatives' practice of setting prices, which most consumers consider to be excessive, also hampers development. (Source: IFR-82/66.)

Canadian Fish Exports Jump Sharply in 1981; Imports Rise Slightly

Canada exported a record $1.24 billion worth of fishery products in 1981, a 24 percent increase over the $1.0 billion exported in 1980. That made it the largest exporter of fisheries products in 1981 and the United States was its largest customer, purchasing $668 million worth of fishery products, 16 percent more than in 1980 when U.S. imports from Canada totaled $576 million.

The European Community was Canada's second largest customer, buying $253 million worth of fishery products in 1981. This was a decline of 4 percent from the 1980 total of $264 million.

Canada exported $103 million worth of frozen blocks in 1981, an 18 percent decrease from the $122 million in exports in 1980. Cod block exports amounted to $83 million in 1981, with the United States buying $72 million worth, a 23 percent decrease from the $89 million that U.S. importers spent on Canadian frozen cod blocks in 1980.

Canada also exported $115 million worth of salted and dried groundfish in 1981, or 35 percent more than in 1980 when such exports amounted to $85 million. Cod products accounted for $93 million of the total salted and dried fishery exports. Portugal was Canada's largest customer for salted and dried cod, purchasing $31 million worth in 1981, more than double the $13 million worth shipped to Portugal in 1980. The United States bought $22 million worth of these products from Canada in 1981, a 15 percent increase over the $19 million spent in 1980.

Canada exported $248 million worth of fresh and frozen shellfish products in 1981, a 5 percent increase over the $236 million exported in 1980. Scallops accounted for $86 million worth of shellfish exports in 1981, with almost the entire amount going to the United States.

Canada exported $114 million worth of canned fish in 1981, up 34 percent from the $85 million exported in 1980. Canned salmon exports were valued at $86 million in 1981, with $47 million worth being exported to the United Kingdom.

Canada imported 129,700 metric tons (t) of fishery products worth $307 million in 1981, a slight increase over the $302 million worth of fishery products imported in 1980. Canada bought $193 million worth of fishery products from the United States, a $7 million decrease from the $200 million spent on U.S. fishery products in 1980.

Canadians bought 9,070 t of shrimp worth $69 million from foreign suppliers in 1981, a 9 percent increase over the $63 million worth of shrimp imports in 1980. U.S. suppliers sold $46 million worth of shrimp products to Canada in 1981, a 16 percent increase over the $39 million sold them in 1980. Mexican shrimp exports to Canada decreased by 9 percent from 579 t (worth $6.3 million) in 1980 to 511 t (worth $5.7 million) in 1981. (Source: IFR-82.)

Mazatlan: A New Mexican Tuna Port

The port of Ensenada has dominated the Mexican tuna industry from its inception. However, Mexico is now creating a modern new tuna port to the south in Mazatlan. The transition of Mazatlan to an important tuna port is being carried out primarily by the large state-owned corporation, Productos Pesqueros Mexicanos (PPM)[1] which has based its Operation Tuna in Mazatlan. Private investors, both Mexican and foreign, however, are also involved.

Tuna was first landed in Mazatlan in 1974. The Mexican-Italian joint venture, Productos Alimenticios del Mar (Palmar), landed 500 metric tons (t) in 1974. Palmar had trouble selling it to local processing plants, however, and kept it in cold stores for over a year. During this time, 300 t of the original 500 t spoiled and had to be discarded. Finally, Palmar gave up on selling the tuna in Mazatlan and shipped the remaining 200 t to Ensenada in 1975. After that experience, Mexican tuna fishermen avoided Mazatlan.

Tuna landings were not resumed at Mazatlan until 1979 when Productos Pesqueros de Mazatlan (Propemaz), the local PPM affiliate, initiated its program to turn Mazatlan into a major tuna port. Landings totaled only 200 t in 1979, but by 1981 they had increased to 13,600 t, or about 20 percent of Mexico's total yellowfin and skipjack catch of 69,000 t.

Propemaz has faced great difficulties in rapidly increasing landings at Mazatlan. Dock workers had no experience in handling tuna. This resulted in damage to the tuna and created delays in unloading the seiners. Propemaz claims that an average of about 150 t of tuna per day could be unloaded (some observers claim that unloading is much slower), about half of the average unloaded at Ensenada. In practice, however, much less is often unloaded. Cold stores are full of tuna and local authorities often only unload what canners can process on any given day. Other difficulties are faced by Propemaz. The existing fishery piers, designed for shallow-draft shrimp trawlers, are not able to handle large tuna seiners which have to use Mazatlan's general cargo docks. Port authorities, however, give tuna seiners low priority, and long delays in unloading the vessels are common. It has taken over 3 weeks to unload some seiners.

Mazatlan's current canning capacity only totals about 65 t per 8-hour shift. Propemaz is able to can about 25 t per shift and the two PPM canners nearby at La Reforma and Escuinapa, about 20 t each. The existing cannery at Topolobampo, located about 200 km north of Mazatlan, adds about 40 t per shift to the canning capacity in the Mazatlan area (Table 1). PPM is building a large new cannery at Topolobampo which, until a new cannery is built by Propemaz, will process tuna unloaded in Mazatlan.

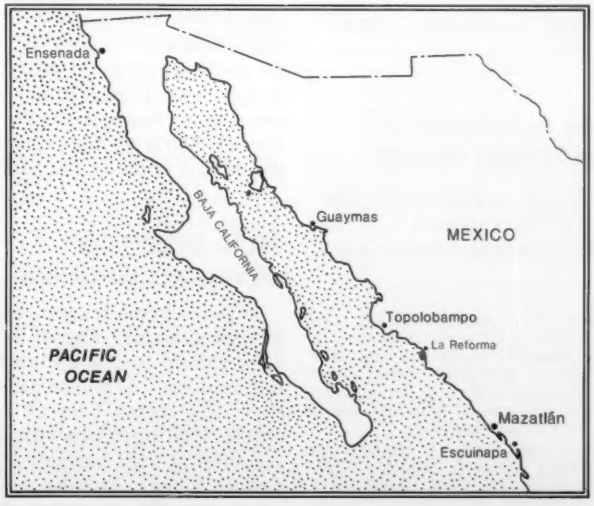
Cold stores in Mazatlan were not able to handle all the tuna landed in 1981. The total cold storage capacity in the Mazatlan area is about 7,100 t (Table 1). Not all of this, however, is available for tuna. Only about 1,500 t of Propemaz's cold storage capacity, for example, can be used for tuna.

Table 1.—Capacity of canneries processing tuna landed in Mazatlan and cold stores at Mazatlan, January 1982.

Company	Cannery cap.[1]	Cold stores cap.[2]
Propemaz	25	3,000
PPM-Escuinapa[3]	20	600
PPM-La Reforma	20	
PPM-Topolobampo	40	
Andsa		3,500
Total	105	7,100

[1]Short tons per 8-hour shift.
[2]Quantity in metric tons.
[3]Located about 75 km from Mazatlan.

As a temporary measure, PPM leased three refrigerated Spanish cargo vessels to store the tuna: *Sierra Fria, Sierra Aramo,* and *Sierra Aranzazu*[2]. These three Spanish refrigerated cargo vessels have added 6,000 t of storage capacity, all of which is being used for tuna. These vessels, however, were not designed to store fish and large amounts of tuna have reportedly spoiled because of improper handling. In addition, repair facilities and spare parts for the vessels are limited in Mazatlan. One of the three cargo vessels has reportedly had to return to Spain for repairs. As needed,



[1]Mention of trade names or commercial firms does not imply endorsement by the National Marine Fisheries Service, NOAA.

[2]The vessels have mostly been docked in Mazatlan; one of the three may have been deployed to Guaymas for a brief period according to an unconfirmed report.

Mexican fishermen have been able to use the new 3,000 t cold store which was completed at Topolobampo in late 1981.

PPM officials insist that the tuna landed in Mazatlan will be canned, primarily for the domestic market. They point out that until the United States embargoed tuna in 1980[3], most of the tuna landed in Ensenada was simply unloaded and trucked across the border to canneries in southern California. PPM maintains that canning the tuna in Mexico creates jobs and increases the food available to Mexican consumers.

Propemaz officials see three advantages in making Mazatlan a major tuna port. First, much of Mexico's tuna is taken at the mouth of the Gulf of California, placing Mazatlan much closer to the fishing grounds than Ensenada. Second, Mazatlan is much closer to Mexican population centers where the tuna is to be marketed. Third, Mazatlan has better facilities to service tuna vessels than Ensenada[4]. Many seiners based in Ensenada have had to use U.S. shipyards.

Propemaz officials are working to solve the existing difficulties. In the future, tuna seiners will land their catch at docks to be located along Mazatlan's new industrial development area, Parque Industrial Bonafil. Propemaz, with Mexican and French investors, has decided to build a large cannery at Bonafil. Private investors are building a cold store. (Source: IFR-82/81.)

Norway Sees Basis for Strong Fishery Growth

It is possible to achieve a considerable and profitable growth in Norwegian fisheries industry, asserts a government-appointed committee which has presented its report to Norway's Minister of Fisheries Thor Listau.

The committee proposes, among other things, a better exploitation of nontraditional types of fish by-products and "waste," and also that more emphasis must be placed on quality. Marketing, product adjustment, and process development should be stimulated, and education and training should be given priority, the report states.

A recommendation was also put forward for a grant of US$16.6 million towards a special readjustment and renewal fund for the industry. The resources will mainly be used within firms and for personnel measures. After a 10-year development program has been implemented, the industry should have a substantially better economic basis for self-sufficiency, on a par with other industries, says the committee.

It was pointed out in the report that three-fourths of the present Norwegian catch is used for fish meal and oil, and that more of the catch should be used for food. So-called inedible fish and other, up-to-now, largely disregarded resources, should be better utilized. On the long term, there appears to be a possibility of a considerable increase in the sales value by also going in for a more planned fishing, the report concludes.

The committee is confident of growth in the fishing industry if its advice is followed and does not rule out the possibility that this industry can, in the future, provide profitable and meaningful employment for more than the 50,000 persons at present employed in it. (Source: Norinform.)

INDIA SEEKS TO DEVELOP FISHERIES

India is reportedly eager to develop its fishing industry. One of its immediate goals is to step up fishery production from 1.69 million metric tons (t) to 1.86 million t for the 1982-83 season. It hopes to attain this goal by encouraging joint ventures and foreign investments in India, two areas which were hitherto eschewed in India's policy.

Also, Indian companies are increasingly going to be authorized to charter foreign vessels. In the effort to improve Indian fisheries, significant financial aid will be granted to assist in building boats, along with other installations, to facilitate importing the necessary construction materials. The government also has plans to lighten the financial load of the fishermen, especially in light of the rising cost of fuel. Thus it appears that the Indian government is making a concerted effort to improve its fishery industry and to promote its exports to Australia, Canada, Japan, the United States, and western Europe. (Source: LSB-11-82.)

France to Modernize Its Fishing Fleet

French Government has announced its new Five Year Fishery Promotion Plan 1982-86, which includes a budget of 2.6 billion francs[1] to modernize its fishery fleet. Of the 2.6 billion francs, 1.4 billion francs will be used for large-scale fishery corporations and 1.2 billion francs will be used for middle- and small-scale fishery corporations.

The plan aims to increase the French catch by 10 percent by 1986. The planners expect French vessels to operate freely within English waters by late 1982. According to the plan, the development of the fishery fleet will be facilitated by spending 5 billion francs to deliver 12 tuna vessels. Also, 1.85 billion francs will be spent to purchase four new distant-water fishery vessels to be used in waters off Canada, Norway, Greenland, and Antarctica.

The Government will be granting subsidies to investments made by fishery firms and will be providing low-interest loans to middle- and small-scale firms. Furthermore, the Government will grant special subsidies to firms which scrap their old vessels to build new vessels. The Government has promised to guarantee the price increase of diesel oil to be lower than the consumer price index. (Source: LSB-9-82.)

[3]The United States embargoed Mexican tuna and tuna products because Mexico seized U.S. tuna purse seiners for fishing tuna within their claimed 200-mile Exclusive Economic Zone (EEZ). The United States does not recognize the jurisdiction of coastal states over highly migratory species such as tuna.

[4]Ship maintainance facilities in Ensenada are currently being expanded. Astilleros Rodriquez is building a major new facility with a syncrolift that will be able to service up to eight large seiners at one time. The new facility should be open by July 1982.

[1]US$1.00 = 6.081 French Francs.

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